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# **SURGERY**

BY

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*FIRST EDITION*

## **VOLUME III SURGERY AND PATHOLOGY OF GROWTHS**



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## PREFACE.

This is the third of the series of eight volumes of Surgery, the whole plan of which is described in the preface of the first volume. It contains the description of all pathological Regenerations and New Formations. The book is intended for those mid-senior students who have just finished a course of Pathology, and are attending Surgery classes.

It is written to the standard of qualifying examinations in England. Being a student of Edinburgh I have mainly followed the teachings of the Edinburgh School of Medicine where I attend the ordinary classes even now when opportunity offers itself. In fact many pages are written from the lecture notes taken by me in the University classes very recently.

In England it is believed that cancer is increasing, and some surgeons there are of opinion that the increased figure is to a certain extent responsible to better diagnosis. Both these factors are true in India, as well, and therefore the study of tumours is becoming important in India as it is in the West.

The classification of some growths, which constitute the border land of inflammatory or reactive blastomatosis or in other words condition of new cell formation on the one hand, and true

blastoma or new growths on the other, in a series of systematic description according to tissues, under a separate title of BLASTOMATOIDS, is a new attempt; and the author hopes that it will give a very clear and comprehensive idea of how one imperceptibly merges into the other at both ends, and thus help the student to grasp the theory of growth more easily.

The same principle of adding a summary at the end of each chapter and marginal notes beside each paragraph has been followed in this volume also as in other volumes with a view to help the student to revise the subject as often as possible at the least expense of time. Those lecturers also who do not find much time at their disposal to compile their lecture notes will find in these summaries a ready made series of hints, which may be useful to them on some hurried occasions.

The Tables set out after genealogical charts are intended to demonstrate the lesions which are associated with each other in common origin and relationship. I have found them useful as a help to remember the broad features of the diseases both as a student as well as a teacher.

The bold types are used in the substance of the paragraph to help the student to find out the main point in the quickest way and thus to relieve the brain from much hunting about for it.

The plates are made from the photographs of the author's own cases. The microscopical

sections of the growths are not included for the simple reason that this book is intended to help the beginner to study the subject from the view point of clinical observations of a growth as it appears when the patient is seen first.

I beg gratefully to thank the authors of various countries of the present day from whose books I have compiled, copied, and quoted freely. The writing of a series of surgery as the present one is a new attempt in India and the difficulties are great, which are so well-known to my readers that they need not be described here. And therefore mistakes and drawbacks are many, for which the author is ever ready to thankfully receive suggestions from his readers.

S. K. SEN.

Burdwan,  
The 17th November, 1930.



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# SURGERY AND PATHOLOGY OF GROWTHS.

## CHAPTER I

### HYPERTROPHY AND BLASTOMATOIDS

#### GENERAL REMARKS.—

If we happen to describe an abscess merely by stating that it is a **swelling**, we may not be far from the truth, and even then we may not be wrong. But except the idea of a swelling such a definition would carry no meaning, and would be very vague for our purpose ; as a swelling means a **tumour**, since the derivative of the very term tumour means a swelling. But the two conditions, that is an abscess and a tumour, are different from each other, although clinically any swelling which we cannot exclude to be an abscess may be described as a tumour.

What  
is a  
Swelling?

Swelling  
means a  
Tumour?

But our trouble to conceive a correct idea of a tumour is not ended here, rather not minimized in the least by excluding an acute abscess only. We know that an abscess is a swelling caused by some infection and consists of a cavity containing purulent fluid. But we may call a cold abscess a tumour, a granuloma a tumour, a fibromatous mass a tumour, a thickened multiplication of some tissues a tumour, a cyst a tumour, and we always describe a goitre as a tumour, a big growth as a tumour, and even to add further, we sometimes describe a gravid uterus of a pregnant woman as a tumour ; and even if we are in doubt in our diagnosis in the case of an acute abscess we are justified to describe it clinically as a tumour. Because, even if a swelling is painful it may or may not be an

But what  
is a  
Tumour?

abscess, and as a matter of fact **any** tumour may become painful by some kind of irritation producing inflammation. We describe a gumma as a syphiloma that is to say a syphilitic tumour.

Clinically  
Tumour  
means  
nothing  
but a  
swelling.

Tumour therefore is an abnormal swelling, and **clinically** the term gives in our mind no other pathological significance but a *swelling*.

Pathologi-  
cally it  
signifies  
a new  
Growth.

**PATHOLOGICALLY** on the other hand the term **tumour** signifies a **growth**, or rather a *New Growth*; or still more clearly it distinctly connotes some new formation of tissues, or formation of some new abnormal tissues which may or may not be quite unlike that of the matrix.

But Hyper-  
trophy is  
also a new  
growth.

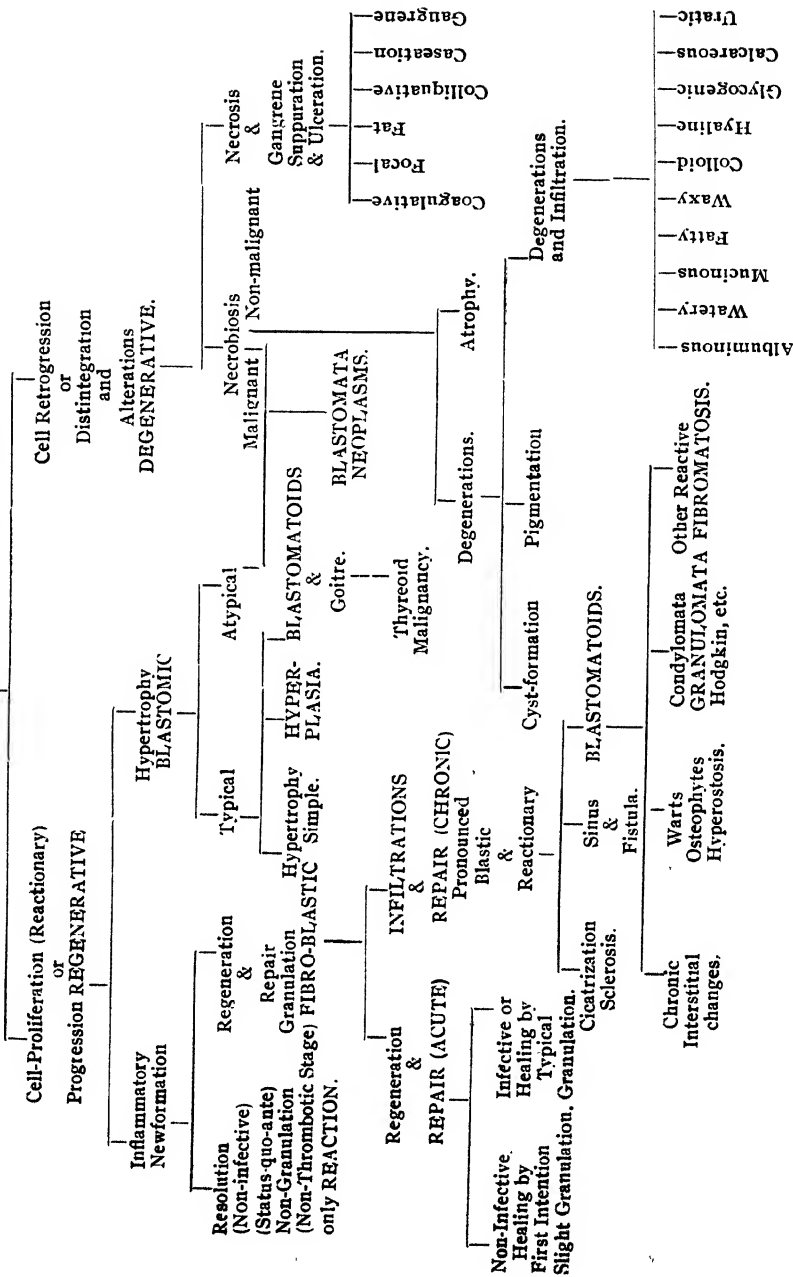
In regeneration of tissues, we find how **new** tissues, and multiplication of the **same** or **new** addition of tissues similar to the matrix take place in some new growths, such as Hypertrophy, or Hyperplasia. We of course do not understand by Hypertrophy or Hyperplasia the same kind of new growth as we do by sarcoma, or by carcinoma, which are described in the proper sense of the term as **new growths**. A mere glance at the Table No. X (facing page 2), will show that Neoplasm is a growth which has both a progressive as well as a retrogressive phase, and it consists of both the processes of cell proliferation as well as an absence of any tendency to control such proliferation, and a presence of a tendency to central degenerative changes and disintegration of cells and tissues.

We have learnt that inflammation is a reaction which is the effect of **irritation**. This reaction is *protective* inducing leucocytosis, *adaptive* with reference to itself and its surrounding tissues, and *reparative* or inducing regeneration. When this regeneration stops short just up to the recoupment of the exact amount of the tissue lost, the process **should** cease, leaving the defect to practically *nil*. If the irritation ceases, the regenerative proliferation should cease as well; but as long as the

TABLE No. X.

## THE EFFECTS OF INFLAMMATION AND IRRITATION.

Facing Page 2.





irritation persists inflammation continues, and regeneration of **fibro-blastic** nature also continues. Thus, if we now consult the Table No. X again we find, that the **growing** tissues may form into a **mass** or a **swelling** till : (i) the irritation may cease, and followed by the *cessation* of further proliferation leaving some abnormal excess, what we call **Hypertrophy**; (ii) the growth may continue to develop and progressively increase in size producing **blastomatoids**, without showing **any** sign of cessation; (iii) the growth may become **autonomous**, that is to say, **every** cell becoming self-proliferating without any control with evidence of other degenerations, which is described as **blastoma**, or **neoblastic** tumour.

So that, it is easy to understand how the same factor **irritation** produces a similar reactive stimulus, and excites **cells proliferation in fibro-blastic, hypertrophic and neoblastic** growths, resulting in :

- A. FIBRO-BLASTIC REPAIR.
- B. EXCESSIVE INFILTRATION,  
AND CICATRIZATION.
- C. HYPERTROPHY.
- D. BLASTOMATOIDS.
- E. BLASTOMA.

But, we know that the different phases, *viz.*, (1) simple **reaction** to inflammation, or (2) **regeneration** and **repair**, or (3) **infiltration** inducing **fibrosis**, or **fibromatosis**, are all the different forms of progressive metamorphosis of **cell proliferation** of a controlled **formative** process and not uncontrolled and **autonomous** as the **blastomata** are.

All are Cell  
Prolifera-  
tions.

It may thus be noted in the said Table that the growth of cells and tissues in a **blastomata** is partly a form of Hypertrophy, but the formation is **atypical**. There is of course no difference in the process of degenerations that any growth or **blastomata** may exhibit or undergo. But we ought to explain what we mean by **atypical**. For the present, let us be satisfied with a simple

statement that whatever is not **typical** or near the standard or **type** means **atypical**. Further explanation will follow.

Pathology of  
Progressive  
Overgrowth. It will therefore be easy for us to understand the pathology and surgery of **blastomatoids** and **blastomata**, if we study the pathology of **progressive overgrowths** in **Hypertrophy** in greater detail. What is Hypertrophy? How is it formed?

## HYPERTROPHY.

ÆTIOLOGY OF HYPERTROPHY.—Tissues are continually and constantly building and wasting. Old cells “yieldeth giving place to new”. The wasting may be due to disease or injury or ordinary katabolism. After injury or inflammation, regeneration is one of the phases of repair by which the new cells are formed. This regeneration is a progressive change. Now, if this progressive change goes beyond what is normally required or the new formation of the tissue be in excess of its normal physiological construction, structure, or form, it is **Hypertrophy**. The original and complicated tissue, if it is of a highly organized nature, may in some cases be replaced by a simpler tissue. It must be particularly remembered, that **epithelium** is the most organized tissue in the body and is constantly being renewed throughout the whole life. At the same time it is more ready than the other kinds of tissues to take on abnormal growth in response to external irritations. This growth may be so perverted as to get out of control of the organism.

Now, let us see and examine the circumstances in which such hypertrophic growths are the actual results. Whatever may be the *immediate cause of producing the hypertrophy, the essential factors and conditions exciting such a growth are :—*

1. Increased blood supply or supply of increased nutrition.
2. The possession of the formative power. In the young, we find more formative power present and therefore, cell division in the young is more pronounced and prompt. Other conditions being equal, hypertrophy is more common in the young than in the adult or aged.
3. The existence of some irritating factor of the nature of inflammatory, or neurotic, or hormonal, or mechanical, or toxæmic, character.

Essential  
factors to  
produce  
Hypertrophy.

Different causes or conditions where Hypertrophy is excited.

**CAUSES OF HYPERTROPHY.**—When we consider about the causes of hypertrophy or rather, we should say, the conditions in which hypertrophy is excited, we may at the very outset observe three prominent different features in them, *viz.*: (a) In some, the tissue element is physiological, the cause of the growth is physiological, and the cells produced are histologically normal. (b) Others where the tissue element is histologically normal, but the growth is produced by, or excited by, some pathological cause pushed to pathological standard. (c) Lastly, we notice some pathological hypertrophy, excited by pathological causes. We shall describe them in the same order in greater details.

I. Cause is physiological and the effect normal.

I. Where the tissue elements of the hypertrophy is **histologically** normal, and the growth is caused by some **physiological** factors. These are the following:—

(1) Internal Secretion.

(1) Hypertrophy caused by **internal secretion** or **hormonic** influence, where the result is carried to physiological standard and the effect of which is meant for some physiological purpose, *e.g.*, development of the mamma during pregnancy is not a pathological growth, nor does it serve any pathological purpose. It is caused by the stimulation of the internal secretion of the placenta called 'placentin'. Its development or hypertrophic condition which it adopts during nursing is due to a separate factor which is described below. Puberty-Goitre is a similar condition of physiological change of puberty.

(2) Inactivity.

(2) Hypertrophy caused by **inactivity** and protection from wear and tear, *e.g.*, the Chinese woman's nails, nails of bed-ridden patients of long standing, the enlarged hoofs of animals, when the legs are not used.

(3) Increased blood supply.

(3) Hypertrophy may be caused by **increased blood supply**, by receiving excessive nutrition, excited by excessive work; thus the causative factor being absolutely physiological, *e.g.*, mamma during the period of nursing, biceps muscles of a hammer man by the constant use of his hammer, increase of the girth of a limb by mas-

sage, pregnant or gravid uterus, keratinized and hardened palms and soles from rough hand-work or walking without shoes, callosities in talipes equinus, or all callosities and corns, hammer-toe, etc., are all instances of hypertrophy caused by excessive work inducing excessive supply of blood, some helped by friction. Grafted nails on a cock's plumes have been demonstrated to have hypertrophied by excessive blood supply. Scapula or other bones of a very muscular male subject in comparison to the standard of those of an ordinary female, are hypertrophied by excessive work.

(4) Disturbance of **hormonic** influence. Hypertrophy in these cases is caused by the overgrowth of the physiological tissue, but the effect is pathological, being pushed to a disproportion or to a pathological condition, *e.g.*, Acromegaly which is caused by the influence of the internal secretions from pathological pituitary body is an example. Hypertrophy of one or two individual fingers of a new-born babe is also an instance of disturbed hormonal condition. The causative factor in such cases is not exactly decided, and is yet unknown. It may be due to faulty hormonal influence or faulty cell division. An interesting specimen of having large 3rd and 4th fingers grown to the sizes of those of an adult in an infant during intra-uterine life, is exhibited in the R. C. S. Museum in London. Specimens are also available of having one or two teeth of a rabbit hypertrophied out of proportion to the others, and the cause of such a condition appears to be the same as of the instances described above.

(4) Hormonic disturbance.

(5) Faulty result of **embryonic** arrangement is another cause, such as the existence of one hypertrophied kidney in place of two normal kidneys.

(5) Faulty embryonic arrangement.

II. **PHYSIOLOGICAL** Hypertrophy from pathological causes:—

The next group of the cases of Hypertrophy are produced by **physiological** infiltration, or nutrition supplied by nature to meet excessive demand, that is to say, factors involving both increased use, producing

II. Physiological hypertrophy from pathological causes.

increased blood supply, and consequently inducing increased nutrition, but **excited by some pathological** factors, such as irritation, inflammation, injury, infection, etc. In this case also as in the previous one, the tissue elements are of normal standard. *E.g.* :—

(1) Septic inflammation.

(1) Infective **inflammation** may produce hypertrophied condition of the bones; for instance, in the osteitic growth from osteomyelitis, the proliferation of bone tissue may be pushed to the conditions of osteosclerosis, hyperostosis, etc. (*Vide* Volume IV). Thickening of the ribs in cases of empyema is another instance.

(2) Irritation of foreign body.

(2) Such hypertrophy is also met with in the bones, produced by **irritation** caused by foreign body, implanted into the tissues as is done in the practice of putting in *gool* in India in which a small log of *neem* wood is pushed in a wound for the treatment of chronic osteomyelitis. Irritation produces hypertrophied condition of the upper margin of the scapula and the clavicle of the Indian *palki bearers* inducing hypertrophied condition of the soft tissues and exciting the development of adventitious bursæ.

(3) Infiltration due to overproduction.

(3) Hypertrophied condition of the abdominal parietes is caused by fat **infiltration** amongst lazy moneyed men in India, which is due to **overproduction**.

(4) Excessive work compensatory to Strain.

(4) Excessive **work** compensatory to **strain** produces hypertrophy in a normal area of a bone, exhibiting no evidence of the presence of any pathological factor; *e.g.*, hypertrophy of the fibula developed after a long standing fracture of the tibia. This happens in other connective and muscular tissues also; *e.g.*, thickening of the capsular ligament of the hip after intra-capsular fracture of the head of the femur, hypertrophy of the heart in Bright's disease, or in mitral disease due to resistance of circulation at the periphery, caused by blood pressure, or incompetence of the valves, exciting more work, or compensatory hypertrophy of the heart in liver diseases. In blood vessels, hypertrophy is produced by mechanical resistance caused by ligation, and the effect of collateral

circulation due to the blockage of the flow. In endarteritis obliterans also the above factors may cause hypertrophy. In the bladder muscles and the ureters, hypertrophy is produced by the obstruction in front, caused by stricture in the urethra, or sometimes idiopathic incoordination of the sphincter nerves, or over-sensitiveness of the sphincters, or under-sensitiveness of the distensers. Lodgment of calculi in the bladder irritates the muscles to hypertrophic condition.

In the kidney, hypertrophy is produced to compensate the absence or death of the fellow kidney, caused by congenital defect or disease.

(5) Excessive **work**, compensatory to **static action**, would produce **hypertrophy** to adjust posture which is influenced by gravitation, *e.g.*, buttress of bone in rickety tibia is a growth for adjustment. (5) Work compensatory to static action.

(6) Excessive **work**, compensatory to discharging the **analogous function** of the co-operating organ, or executing the function of the dual organs, or extra work thrown in, due to suspension of counter secretion, and thus disturbing the harmonic balance, causes hypertrophy of the opposite glands, or of the organs with analogous function, or of the fellow, *e.g.*, removal of the thyroid produces hypertrophy of the pituitary body. Experimental excision of the spleen produces hypertrophy of the mesenteric glands. Removal of one kidney produces hypertrophy of the other, and so forth. (6) Work compensatory to analogous function.

(7) **Irritation** caused by septic **poisons**, as chemical poison, would cause hypertrophy, *e.g.*, hypertrophy of the blood vessel caused by toxæmic poison as alcohol, or the muscular coats irritated by toxins produced in chronic Bright's disease. Coarse black hair grows over tuberculous joint. In varicose ulcers, the hair and the hair follicles in the neighbourhood show evidence of overgrowth. Chronic phosphorous poisoning induces hypertrophy of the bone, and so on. (7) Irritation of poisons.

(8) Only excessive blood supply.

(8) Excessive **blood supply** without the help of any other factor previously described such as irritation, or toxin, will also produce hypertrophy, *e.g.*, in some forms of osteosclerosis, in callous ulcers, growth of abundance of nails in the clubbed hands of patients suffering from mitral disease producing venous stasis, or venous congestion caused by the pressure of a tumour, are similar instances.

(9) Neurotic disturbances.

(9) **Neurotic** disturbance will cause hypertrophy exciting more work, or blood supply, *e.g.*, in the œsophagus, hypertrophy is met with produced by cardiospasm due to inco-ordination of the action of the vagus. In the pylorus of the stomach, hypertrophy may be acquired from obstruction or inflammation. Hypertrophy of the pylorus may be congenital also. Over-sensitiveness of the pyloric mucosa due to a hysterical condition sometimes produces hypertrophy of the pylorus. In the transverse colon, dilatation with hypertrophy is met with produced by hyper-sensitiveness and hypo-sensitiveness. In the pelvic colon, hypertrophy is produced by malaction of the plexus of Auerbach, causing kinking and constriction and sometimes inducing obstruction of the bowels. Hirschsprung's disease is a condition of enormous dilatation of the sigmoid flexure accompanied by hypertrophy.

(10) May be adaptive.

(10) **Adaptive** hypertrophy of the bone is found in some instances, such as the thickening of the skull in senile atrophy of the frontal brain, inducing adaptive hypertrophy of the frontal bone. The hypertrophy here, is uniform in all the tables. Thickening of the skull in epileptic, imbecile, inflammatory osteitis deformans, etc., are other instances of hypertrophy caused under similar conditions.

III. Pathological causes producing pathological hypertrophy.

III. **PATHOLOGICAL** hypertrophy from **pathological** cause. The tissue elements are usually fibroblastic, the cause and the effect being all pathological, *e.g.* :—

(1) Pseudo-hypertrophic muscular-dystrophy, as has already been stated above, is really a hypertrophy of the

fibrous and fatty tissue with actual atrophy of the essential cells, which in this case are the muscular cells.

(2) Hypertrophic cirrhosis of the liver Here, the (2) Cirrho-actual essential cells are atrophied with degenerative<sup>sis</sup>. hypertrophy.

(3) Hypertrophy of the spleen.

(4) Hypertrophic amyloid large kidney.

(5) Hypertrophy in blood-forming organs, *e.g.*, the bone marrow is hypertrophied in :—

(a) Rickets.

(b) Anæmia.

(c) Polycythemia.

### CONGENITAL OR ACQUIRED—INHERITED.

It has been stated that hypertrophy is acquired, but congenital hypertrophy is observed in a few instances. The acquired condition is also inherited, *e.g.*, the buttock of a young ape, or the soles and palms of human babes are such instances.

## BLASTOMATOIDS.

Blastoma-  
toids.

Thus, we understand something of the pathology of **progressive overgrowths**. But in all the instances of such growths as happens in Hypertrophy, we find, there are two elementary factors present, namely, there is a **purpose** and there is a **limit**.

In the blastomatoid conditions, the overgrowths we encounter with, are conditions where there is a **purpose** of reactive reparative nature, but the fibroblasts and proliferated cells do not exhibit any tendency to **limitation, or termination** according to the amount of loss, or do not stop short and remain limited to natural standard. The processes in Blastomatoids are **reactive** and **hyperplastic**; exactly as they are so in infiltration and Hypertrophy on the one hand, and Blastomata, —first benign and then malignant,—on the other. Thus in that sense, the **Blastomatoids** constitute the **link** in between inflammation and tumour. We shall find later on that in the right wing group, that is, the Blastomata with which Blastomatoids exhibit more sympathy and relationship, there is neither any *purpose* nor any *limit*, so far as the progress of the growth is concerned.

The co-  
relations.

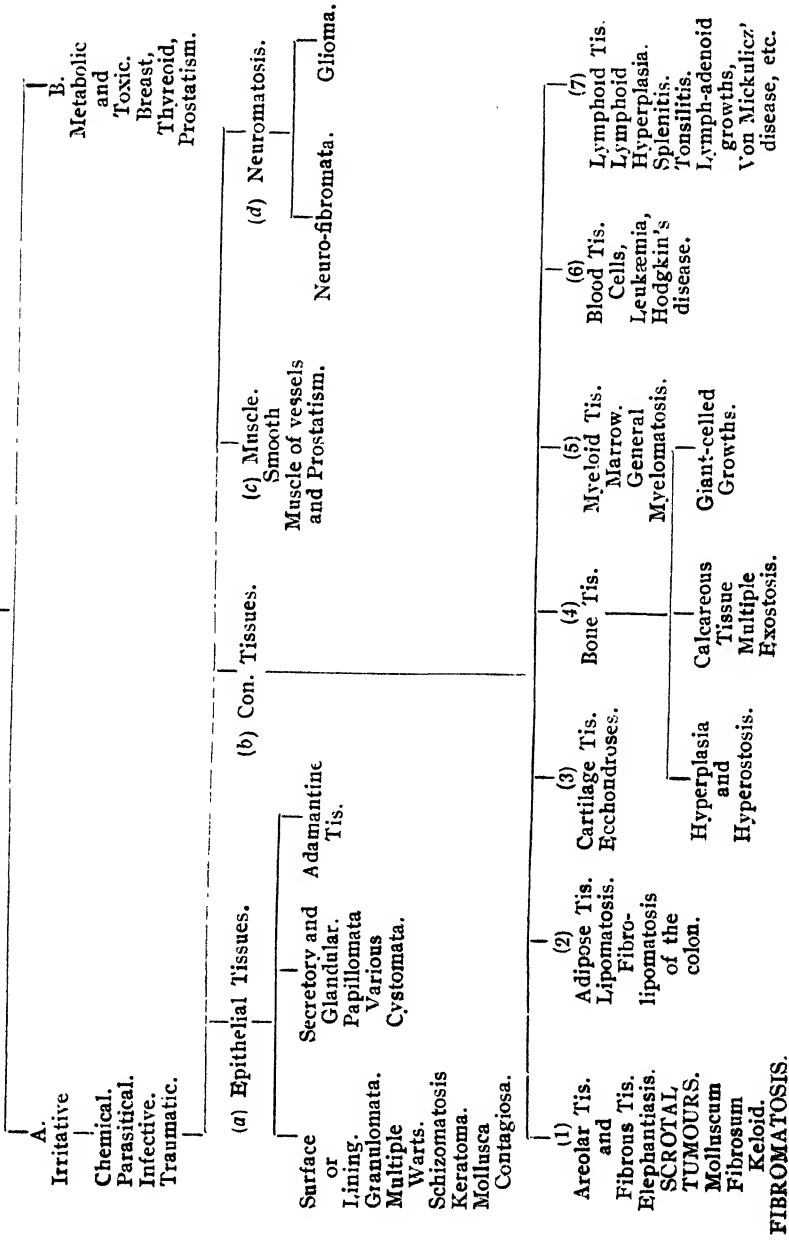
We have shown the co-relations existing between the different progressive Infiltrations, Hypertrophy and Blastomatoids. *Vide* Table page 2. The different types of Blastomatoids met with are set out in a table below :—

As Blastomatoids are reactive and hyperplastic processes, growing as a result of the reactions or effects of inflammation the grouping of such growths produced by blastomatoid process may be done under the same headings as the factors which cause inflammation.

It is not possible to describe all the Blastomatoids in detail here, for which the reader is referred to Regional Surgery. We shall simply discuss some general pathological principles detailing some well-known instances at this

Table No. XVIII.

## BLASTOMATOIDS.



FIBROMATOSIS.

stage so that it becomes easier for the reader to follow **Blastomata**.

It has already been stated, in describing the final stage of fibroblastic repair or overgrowth of various different tissues, that the development of a Blastomatoid takes place as a further extension of the hyperplastic and reactive process; the overgrowth retaining its nutrition and growth from the new blood supply it received from the young vessels during the formation of granulation which is never absorbed as happens in an ordinary cicatrix. The origin and cause of the hyperplastic and reactive process of which may be :—

A. IRRITATIVE, including under it the chemical, parasitical, infective and traumatic causes.

B. METABOLIC or Toxic.

*vide* Table XVIII.

We shall proceed to explain further :

A. IRRITATIVE—

A. Irrita-  
tive.  
(a) Chemi-  
cal.

(a) *Chemical*.—Chemical substances such as paraffin, if handled for any length of time, will produce a condition of chronic irritation resulting in hyperplastic overgrowth of tissues. The condition may remain of the nature of simple granulation fibrosis for sometime; but in most instances it gradually passes on to blastomatoid condition, a further stage of which is frank Carcinoma or Blastoma.

(b) Parasit-  
tical.

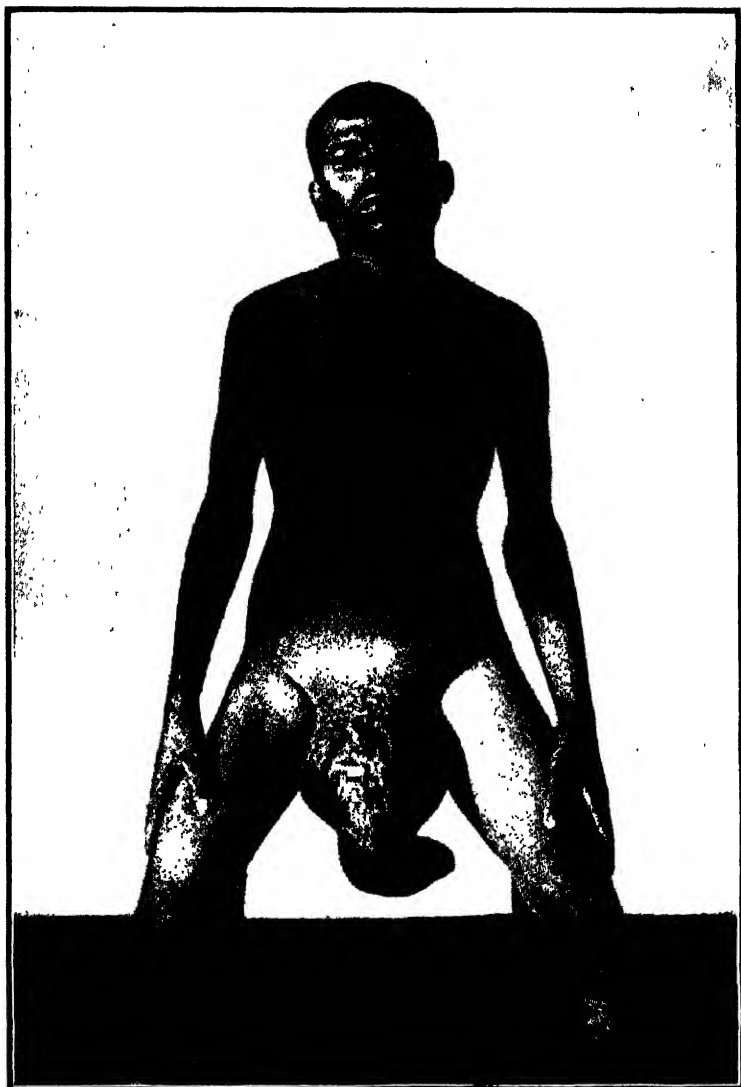
(b) *Parasitical*.—Coccidiosis in rabbit and Bilharziasis or Schizomatosis in man are striking examples. The condition of Schizomatosis is a papillomatous hyperplasia of the mucosa of the bladder and rectum, scarcely to be distinguished from Blastoma ultimately ending in Carcinoma. For further analysis of blastomatoid conditions of the skin or mucosa *vide* chapter on Non-malignant Blastomata.

(c) *Infective and Traumatic*.—

(c) Infec-  
tive.

Like simple irritation as described above, irritation from infection may similarly give rise to Blastomatoids.





ELEPHANTIASIS OF PENIS.

The different examples are classified according to different tissues particularly those which take the most prominent part to exhibit the tendency to hyperplastic proliferation. Those are as follows :—

(a) **EPITHELIAL.**

(a) Epithelial.

This may again be divided into:—

(1) Those arising from the lining or the surface type of epithelium.

(2) Those arising from the secretory or the glandular type of epithelium.

(1) *Surface type.*—The different examples are:— (1) Surface Type.

Granulomata,

Condylomata,

Papillomata,

Multiple Warts.

The reader will easily realize his danger of stepping further down ; as he knows well that his next step would mean a drift beyond his depth,—a limitless, fathomless, fountain of proliferation,—or in other words, **Carcinoma** or **Epithelioma**.

(2) *Glandular Types*:—The various examples are the Cystomata described in the last volume, and the other examples are Cyst-adenomata to be described. *Vide* Chapter III. We are afraid we shall find ourselves confined in the cystic chambers of death if we proceed any further. The **Adenocarcinomata** would be found blinking before us !

(2) Glandular Type.  
Cystomata.  
Cyst-adenomata.

Other blastomatoid processes of the surface epithelium are :—

**MOLLUSCUM CONTAGIOSUM** is a multiple blastomatoid process which arises in connection with the epithelial cells of the skin. In all probability, the process is an infective one and should always be regarded as such.

Molluscum Contagiosum.

The tumours which are situated immediately beneath the epidermis are of multiple nature, each of which consists of a nodule with a slight **umbilication**. A nodule

histologically represents a growth constituted of solid alveoli of epithelial cells separated from one another by processes of connective tissue, all these being arranged around a central point which is represented by the umbilication on the surface. The cells of the most inner circle exhibit an anomalous **keratinisation** as the result of degeneration. The individual degenerated cells are histologically described as **molluscum bodies**. Degeneration of many cells at the centre of the tumour produces a softness of its consistence, such an area being completely occupied by the degenerated cell debris underneath the surface.

CLINICALLY it is usually seen amongst children, and the condition often passes from one child to another. The size of a nodule does not generally become bigger than a pea. The distribution is particularly observed on the face, lips, and eye lids.

TREATMENT consists in thorough excision followed by curetting and cauterizing the area with strong caustics.

### BLASTOMATOIDS OF DENTAL EPITHELIUM.

Blastomato-  
toids of  
Dental  
Epithelium.

The type of epithelial blastomatoid process, seen in connection with the teeth, especially the dental sac and its contents, as well as the neighbouring bone, are fairly common. These blastomatoid processes and some blastomata are included in one group of growths clinically described as **odontome**. The reader will understand them better after he finishes his observations on blastomic conditions of the teeth, described under Dental Exostosis, and in the chapter on blastoma.

(b) Connective  
Tissues.

### (b) CONNECTIVE TISSUES.

(1) Areolar  
Tissue.

#### (1) Areolar tissue and Fibrous tissue:—

Scrotal  
Tumours.

**SCROTAL TUMOUR.**—Examples of Blastomatoids we come across in connection with connective tissues of



SCROTAL TUMOUR.



the skin are best represented by Elephantiasis of the leg and scrotum. Elephantiasis is a mixture of chronic lymphadenoma with superadded infection resulting in fibromatosis. For the details *vide* the regional part of the Surgery. See photo plates Nos. XXXI & XXXII.

**KELOID** is an instance of hard blastomatoid pro- Keloid. cess of the nature of fibromatosis developing in connection with the fibrous tissue of the skin. It is sometimes described as a scar-tumour already mentioned under the description of Scar. *Vide* Volume I. Keloid tumours especially develop from scar tissue on the shoulders, chest, face, etc., and are usually associated with some form of irritation either of infective nature of long standing, or burns. It is also relatively common on the neck of tuberculous patients after repeated attacks of chronic suppuration of tuberculous glands in the area when it peculiarly develops in the transverse direction. It may also develop at the site of any healed wound, which may be quite unimportant or of an insignificant nature. This shows that the patients subject to keloid must have a striking congenital predisposition to the condition.

**MICROSCOPICALLY** a Keloid is composed of fibrous tissue and spindle-cells which in some instances may resemble the structure of a fibro-sarcoma. A Keloid consists of dense bundles of collagen which are often hyaline in appearance between which a few fibroblasts may be seen to lie compressed, although in the early stage keloid tissue may be more cellular and therefore softer. At the periphery of the growth no true capsule can be made out, and a certain amount of cellular reaction around the cutaneous blood vessels of the nature of perivascular **fibrosis** is always observed. Extension of the growth takes place around the vessels of the deeper layers of the skin. Keloids may increase indefinitely or may stop short of growth at any stage, which happens in many instances, when the tumour spontaneously undergoes atrophy wholly or in part. It is often found useless to remove these scar tumours, as

one is succeeded by another tumour of the same kind, as the fibroblasts developing from the operation wound proliferate to form tumours exactly like its predecessor. It may start in even simple wounds by any irritation caused to the cicatrix. Injections of fibrolysin or idolylin may be useful.

Burns are often followed by the development of Keloids especially if treated on wrong lines. *Vide* photo plate No. XXXIII.

Keloid has been reported to have developed from red dye in tatoo, though not from tatoo done by blue.

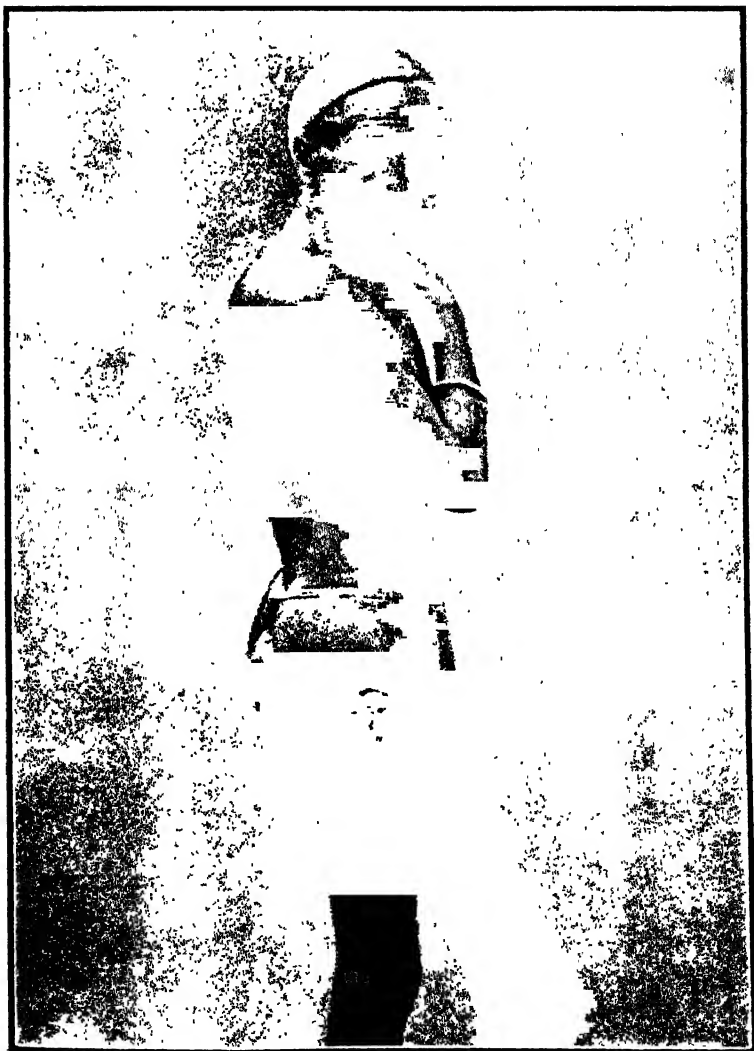
Predisposition to Keloid growths is not only seen in individuals but is seen also in some races; *e.g.*, Negroes are peculiarly liable to develop Keloids.

Molluscum  
Fibrosus.

**MOLLUSCUM FIBROSUM** is a blastomatoid process which arises in connection with the sheaths of the nerve. Although it is composed of fibrous tissue its association with nerve elements is so close that it is clinically described as a nerve-tumour or Neuroma. Molluscum Fibrosus is in reality a growth of blastomatoid nature resembling a fibroma in its histological features. It develops either as a single or multiple smooth-topped freely movable nodule, sometimes in large masses or pendulous folds from the epineurium. The nodular forms may be hard and consist of dense fibrous tissue. For further details the reader is referred to the blastomatoid processes in connection with nerves at the end of this chapter. *Vide* page 41.

Fibromatosis.

**FIBROMATOSIS** are extensively met with in some organs, *e.g.*, the breast, prostate and the kidney. They consist simply of an overgrowth of fibrous tissue retaining their nutrition from young vessels developed in the preceding granulation tissue, the structure of which is closely allied to Fibroma; but having no capsule. The tumour-like nodule fades off into the surrounding tissue. Frequently they are seen in **multiple** forms. Fibromatosis is a slow growing tumour which may contain glandular elements also, the latter being incorporated



KELOID.



into the substance of the mass in the course of its growth. It is **not** a **neoblastic** process and this is the form of growth which is very difficult sometimes to distinguish from true blastomata. Only by very careful examination of the whole growth it is possible to determine the diagnosis. A little section from one part of the tumour under the microscope is of little help to the pathologist.

ALL tissues of the body are subject to similar growths which are briefly mentioned in this chapter. For the details of these processes with reference to particular sites the reader should consult the regional part of the book.

Sometimes, malignant transformation to sarcomatous nature supervenes in late cases of Fibromatosis.

## (2) ADIPOSE TISSUE.

Blastomatoids met with in connection with proliferation of normal fat cells, quite distinct from the subcutaneous and abdominal fatty deposits of obese, working under some stimulus of reactive hyperplastic nature are the following :—

### (i) DIFFUSE SUBCUTANEOUS LIPOMA.

Diffuse Lipoma is a condition of limited localized accumulation of normal fat. This infiltration of fat takes place in the subcutaneous tissues of some particular region quite distinct from such accumulation in the obese. It is always, or almost always, symmetrically distributed. These growths are multiple and not encapsulated. They freely infiltrate and burrow around neighbouring tissues ; sometimes involving important structures.

Men, especially those who are heavy and obese, beer drinkers, and lazy in habits, after the age of forty, are the sufferers from this type of Lipomatosis. Subjects with very heavily doubled chin are the common instances met with as examples of diffuse Lipomatosis. The condition is also well seen in the massive **perirenal Lipoma**.

(i) Diffuse  
Lipoma  
in Men.

Doubled  
chin.  
Peri-  
renal  
Lipoma.

TREATMENT consists in prohibition of all alcoholic drinks and fatty foods. Excision is soon followed by further recurrence and therefore it is useless.

The most important point is its tendency, like malignant growths, to invade, infiltrate, and burrow into the surrounding tissues. As there is no capsule around this diffuse growth, it is not possible to demarcate it from the normal fatty layers. For this reason, recent observers do not consider these growths as non-malignant blastomic tumours as it used to be described before. The infiltration may involve important structures, exerting dangerous pressure on important organs, leading to serious symptoms. This infiltrating tendency of this type of lipomatosis is an exceptional instance of Blastomatoids. Lipomatoses are the results of excessive proliferation of hyperplastic nature of normal fat only.

TREATMENT consists in adopting antifat measures.

### ADIPOSIS DOLOROSA, DERCUM'S DISEASE.

Adiposis  
Dolorosa or  
Dercum's  
Disease.

Like men as happens in the previous condition, women suffer from overgrowths of adipose tissues almost similar in nature to the above; but the growths are *painful* as the name implies and they develop at various parts of the body.

Women at climacteric or after the age of forty are the sufferers from this kind of Lipomatosis. This affection is really due to the effects of hypothyroidism, as manifested by other symptoms of thyroid deficiency.

Similar deposition of subcutaneous fat associated with loss of function or atrophy of the genital organs also occur in some cases where the pituitary body is absent or destroyed. It is sometimes seen as an after effect of Kala-Azar.

Clinical  
Features.

CLINICAL FEATURES. The symptoms and signs are ushered in by the presence of severe pain. The pain is of a neuralgic type, and may precede or associate with the accumulation of fat.

**TREATMENT** consists in Endocrine therapy, regu- Treatment.  
lation of diet and habits. Prolonged administration of  
thyreoid gland, or mixed gland tabloid may be helpful to  
diminish the fat. Bowels must be kept freely open by  
saline purgatives.

If an operation is thought advisable it is better to cut  
through deeply on the mid line of the substance of the  
tumour and then deal with each half shelling the fat out  
from each half of it. An extensive dissection is required  
to separate the fat lobules from their deep attachments.

### 3. BLASTOMATOIDS OF CARTILAGINOUS TISSUE.

ECCHONDROSES are cartilaginous growths of blas- 3. Blastoma-  
tomatoid nature which develop from the ends of long toids of Car-  
bones inside the joint capsule in Osteoarthritis. tilaginous  
Tissue.

Owing to the toxic actions of an unknown causal Ecchon-  
agent, the articular cartilage degenerates, making the drosis.  
smooth surface of the central part of the end of the long  
bone constituting the joint rough. In the peripheral  
part on the other hand active proliferation of the carti-  
lage goes on, succeeding the degenerative changes in the  
central area, resulting in the development of excrescences  
which gives the lateral aspect of the head of the bone  
the appearance of drippings from a half burnt tallow  
candle, sometimes forming a ring around the edge,  
giving it an appearance of **lipping**. These excrescences,  
drippings, and lippings, are called **Ecchondroses**. The  
new cartilage in Ecchondrosis is largely formed by  
the proliferation of the cells of the synovial perichon-  
drium. These Ecchondroses are sometimes described as  
**Chondrophytes**. Parts of Ecchondroses may be detached  
to form the **joint mice**. Many chondrophytes are ossified  
and converted into osteophytes.

For the details regarding other various blastomatoid  
changes in cartilage and modifications in Osteoarthritis,  
and their clinical features and treatment the reader is  
referred to Inflammation in Bone. *Vide* next volume.

4. Osteal  
Tissue.

## 4. BLASTOMATOIDS OF OSTEAL TISSUE.

Various growths of Blastomatoid nature are seen in bone, and clinically so many inflammatory and traumatic conditions associated with new bone formation are met with, that it is sometimes very difficult to state definitely what should be included in the group of Blastoma of bone. By examining the histological features of different cut sections it is not possible to exactly differentiate a blastoma of a bone from callus following a fracture, new bone formation in osteomyelitis, the ossification of inflammatory exudates by infiltration or deposition of calcareous substance, exostosis, myositis ossificans, or osteophytes in arthritis deformans, or as a matter of fact any bone-forming blastomatoid process.

Subungual  
Exostosis.  
Dental  
Exostosis.

**SUBUNGUAL EXOSTOSIS** and **Dental Exostosis**, although described as bone tumours are really blastomatoid processes arising from the direct effect of inflammatory reactions ending only in hyperplastic regeneration.

All these different clinical manifestations of blastomatoid processes of bones require a detailed description to enable us to understand their clinical nature; for which the reader is referred to Inflammation in Bone.

The most important blastomatoid process met with in connection with bone is the **Myeloid** or **Giant-Celled Tumour** as it is called.

Bone primarily consists of, (i) periosteum and endosteum, made of fibrous tissue and osteoblastic and osteoclastic cells; (ii) marrow, consisting of fat cells, marrow-cells or myelocytes; amongst which many erythroblasts or normoblasts, and many osteoclasts, **myeloplaxes** or **megakaryocytes** or **giantcells** are found; (iii) osteum, made of calcareous tissue, a frame of purely inorganic chemical substance. So far as blastomatoid processes in connection with bone are concerned, the most important clinical condition met with is Myeloma. The term Myeloma by itself is unfortunate as it indicates

a growth originating from myeloid or fat tissue which certainly it is not. These tumours are not originated in the marrow.

**A MYELOMA** is not a Blastoma or a neoplasm, or a new growth as we call it; and therefore not a tumour as it is understood by the latter term; but it is a tumour from the sense that it is a swelling. It is of course an inflammatory swelling and not a blastomic one. The most scientific terminology of the condition would be to describe it as **giant-celled blastomatoid growth**. It is called by various other names, *e.g.*, Myeloma, Myeloid Sarcoma, Giant-celled Sarcoma, Benign Giant-celled Tumours, etc. It is a growth of **blastomatoid** nature, *situated* in the bone-marrow, fibroblastic in nature, derived from the same kind of bone reticulum from which normal osteoclasts are formed, but certainly not originated in the marrow itself. Most of these forms were formerly described as Myeloid Sarcoma, but they belong to a distinct type of inflammatory overgrowths which are neither tumours of non-malignant nature, nor sarcomatous conditions in any way. At the very outset, it must be clearly understood, that it is a mistake as well as confusing to call the condition by any other name but **giant-celled blastomatoid growth**. All its adjectives such as benign, myeloid, etc., or its other names giving it a significance of its being a tumour, or to call it a Myeloma having anything to do with marrow or by any term with the suffix **oma** should be discarded.

The characteristic feature of these tumours is the presence of multi-nucleated giant-cells of the nature of foreign body type; but their function is probably destructive and therefore they are regarded by some as Osteoclastic. These giant-cells are not like those found in the tubercle or granulomata. The latter kind of giant-cells have got a regularity of their arrangement, position and structure, and a relationship with the surrounding neighbouring cells. (*Vide* chapter on Chronic Inflammation). These giant-cells on the other hand: (a) vary

Myeloma  
or  
Myeloid  
Sarcoma  
or Giant-  
Celled  
Tumour.

Giant-  
Celled  
Benign  
Tumours.

a good deal in size and shape and appear like groups of foam; (b) the nuclei are oval and more abundant in number, which are generally 15—20 in granulomatous giant-cells; and which in these giant-cells again are not distributed around the periphery, as in the case of tubercle type of giant-cells, but are more disposed towards the centre of the cell; (c) the outline of the giant-cells in these tumours may be regular, but more often is not so, and sometimes is projected out as interlacing processes; (d) the tuberculous giant-cells have a definite concentric arrangement with the neighbouring giant-cells and other kinds of epitheloid, connective tissue, and round-celled cells around them. There is no such definite grouping of cells in these tumours, but as described above appear like groups of foam. The term Myeloma as stated above, is a misnomer and these giant-cells do not arise from the *fat* cells or even their precursors called Myeloplaxes; which are a kind of normal cells of multinucleated type of giant-cells found normally in bone-marrow. These giant-celled blastomatoid growths arise from the connective tissue or reticulum of the periosteum or the endosteum from which the osteoclasts are derived. The giant-cells are of foreign body type called into being through the eroding action of the growth on bone. The tumours are certainly not bone-marrow tumours. In some cases large cells called **xanthoma** cells containing anisotropic fat or doubly refractive lipid probably cholesterol ester are found.

Xanthoma  
Cells.

MICROSCOPICALLY, the growth is composed of matrix consisting of the above giant-cells which are embedded in a large mass of spindle-shaped cells and a few round or oval cells. These giant-cells may be twenty or thirty times bigger in size than the other cellular structures around them. The proliferating edge of a fresh tumour looks dark maroon colour, which on section, exhibits a multicystic nature, and each cyst being filled with blood, gives it an appearance of a cut pomegranate. The growth is very vascular and

reminds us of its nature of granulation tissue; in some cases, pulsation may be perceptible at the most vascular points where the bone is absorbed, yielding the clinical sign of "egg-shell" crackling on gentle pressure. This type of growth has also been described by some authors as **chronic Hæmorrhagic Osteomyelitis**. Some of these cysts are filled with serum and yellowish fibrinous clot; otherwise, the intercellular substance is more of a gelatinous nature. Some grow into a large Bone-cyst as described before. Cyst formation may occur in the centre at the expense of the cancellous tissue which being absorbed turns the wall into a mere shell, which in turn is continually re-inforced by new formation of bone from the periosteum.

In consistency these tumours are soft, and on scraping, a slimy or sero-sanguinous fluid is obtained.

The most important feature is the cystic character of these growths. In fact the very existence of cysts and the allied conditions of the formation of inflammatory new-formations often remain undiagnosed, if done without the help of X-rays, till we understand their real nature under the microscope after amputations, which have often been unnecessarily done; or they become evident by spontaneous or traumatic fractures. Metastases never occurs in these inflammatory tumours.

Some authorities, especially Ely and Barrie, consider these tumours to be similar in nature to **Osteomyelitis Fibrosa**; for further details of which the reader is referred to chapter on Osteitis Fibrosa. *Vide* Vol. IV.

**PATHOLOGICAL TYPES.**—Two main types are met with, namely:—

(1) Periosteal and, (2) Medullary.

(1) The **periosteal** type is seen in the maxillæ, and periosteal giant-celled blastomatoid tumour constitutes the commonest form of **epulis**. It is a growth of firm consistency and of slow progress. It does not attain

Pathological types.

(1) Periosteal types. Epulis.

a large size. For further description *vide* regional surgery.

(2) Medullary type at the ends of the long bones.

(2) The **medullary** type occurs in the long bones at the ends *remote from* the directions of the nutrient vessels; *e.g.*, lower end of the femur and upper end of the tibia, upper end of the humerus, and the lower end of the radius.

Clinical Features.

**CLINICAL FEATURES.**—Giant-celled blastomatoid is not a neoplasm, and does not exhibit any malignant nature as it used to be described formerly. To call it myeloid-sarcoma as stated above is not correct, as that brings an idea of malignancy in it. This growth is always of a localized nature, and its expansion within the bone is strictly limited, with no tendency to diffusion along the medulla. The multiple lesion known as Diffuse Myelomatosis described below, under the blastomatoids in connection with bone-marrow, is quite of a different pathological and clinical nature. This localized type of Myeloma, as this variety of blastomatoid is sometimes mistakenly called, does not give rise to secondary deposits. The lymphatic glands or viscera are not affected. It is generally ramparted by an effective barrier of condensed bone tissue confining the growth into a localized lesion. This latter feature and its non-extension beyond the periosteum, occupying a central position are its characteristics which are clearly visible in radiographical examination. Formation of bone cyst is a very common feature in giant-celled tumours. The tumour is very vascular and consists of all features of young granulation tissue.

Sites.  
Very commonly at the ends of the bones which ossify last.

**SITE OF ELECTION.**—As stated above it generally develops in the later-growing-ends of long bones which ossify last; *e.g.*, upper ends of the humerus and tibia, lower ends of the femur and radius, that is to say, **away** from the directions of all nutrient arteries in the bones; as they are directed towards the ossifying centres which unite first. In this connection, it may be remembered, that these are the very ends of the long bones where union and ossification take place quicker after fractures.

When the growth develops in the periosteum of the lower jaw forming a kind of jaw tumour, it is called **epulis**. These growths also develop in tendon sheaths, bursæ and the capsule of the joints.

Also at the jaw, tendon sheaths, bursæ, capsule.

**CLINICALLY**, swelling is the only characteristic symptom. This swelling is globular, it is not red, and is not hot. It is not cystic to the feel as the cysts develop in the centre of the bone ; but it may give a feel of crackling on pressure described clinically as "egg-shell crackling" ; as we sometimes meet with in Hydatid of bone. The swelling as described above is situated at the long axis of the bone, and is caused by the so-called *expansion of bone*. The expansion is produced by the absorption of the osseous tissue at the inner aspect with the accompanying new bone formation externally from the periosteum. The outer layer by such expansion and distension is gradually thinned out, the laminæ of which yield and break on pressure giving rise to the "egg-shell crackling". As the blastomatoid tumour grows in the central aspect, the *globular* nature and the eccentric character of the growth persist with its expansion ; and sometimes, this expansion and thinning out of the shell go so far that it is surprising to observe how the continuity of the bone could remain intact and not fractured in the meantime.

Signs and Symptoms. Swelling of progressive nature. No redness or heat.

**PAIN** is the next common symptom. The pain is more of a subjective nature, similar to that of chronic osteomyelitis ; and since the onset is similar to that of chronic osteomyelitis the pain should suggest the nature of the lesion to be that of inflammation. In most of the instances this pain may be associated with some kind of trauma. Spontaneous fracture may occur at any time. There is no tenderness.

Pain.

**FRACTURE** of spontaneous nature is often associated with violence of a kind so trivial in nature, and so out of proportion to the extent of injury and damage that the occurrence of such fracture only might preci-

Fracture.

pitate the correct diagnosis of the actual nature of the lesion, or account for the pain persistently complained of, before, by a patient under observation before the fracture took place.

Deformity,  
Disability,  
Muscular  
Atrophy.

DEFORMITY, DISABILITY, AND MUSCULAR ATROPHY may be present in a pronounced case. The neighbouring joint usually remains unaffected, although in an advanced case the growth may extend around the articular cartilage and thus obstruct the free movement of the joint.

RADIOLOGICAL FEATURES.—On radiological examination a well-defined area of bone more translucent than normal, but scattered with dark foci caused by the presence of calcareous masses, is revealed exposing the growth. This X-ray picture of the growth thus exhibits a markedly characteristic multicystic appearance with well-defined and sharp limitation from the soft tissues.

Treatment.

TREATMENT.—It should always be remembered that the growth is not a malignant growth or as a matter of fact not a tumour at all, and the method of simple excision and curetting serves the full purpose of treatment. Amputation becomes necessary where repeated fractures or the size of the growth have turned the limb quite useless. In most of the cases scraping and dealing with the cavity as that of a chronic bone abscess is successful. The cavity left may be filled with fat or muscle grafts.

Exposure to X-ray or radium treatment after scraping may be necessary in the cases where the cleaning could not be done satisfactorily by curettes. In the cases where more than one bone is affected, *e.g.*, both the lower end of the femur and the upper end of the tibia of the same side, amputation may be imperative.

Repeated local irritation in the form of unsuccessful or insufficient curettage has been known to transform the condition into malignant spindle-celled sarcoma.

## 5. BLASTOMATOIDS, IN CONNECTION WITH BONE-MARROW.

In considering the blastomatoid processes in connection with bone it has been described that the growth which used to be described as Myeloid Sarcoma is really not a malignant neoplasm. Such growths therefore was described, especially by English authors, as Myeloid Tumours; and this name was given on the supposition that the growths concerned were blastomic in nature and arise from marrow. Both these features are now proved to be wrong. These tumours are neither blastoma nor do they arise from bone-marrow. Myeloids are simply inflammatory blastomatoid processes arising from the same reticulum of bone from which giant-cells are derived. They are therefore called **giant-celled** tumours. To avoid confusion in the minds of beginners, we have discarded the term tumour also, and called it as **giant-celled blastomatoid growths**. In consideration of the above features the term Myeloma, which is also used to describe a form of Epulis, where a similar-kind of cells like the giant-cells found in the giant-celled blastomatoid growth are present, has also been discarded. And therefore the question of grouping some tumours as Myeloma and some as Diffuse or Multiple Myelomata does not arise. Since Diffuse Myelomata actually arise in the bone-marrow, this condition only has the right to be called Myelomata, or Myeloma, and this multiple or diffuse condition only deserves this designation.

(5) Blastomatoids in connection with Bone-Marrow

**MULTIPLE OR DIFFUSE MYLOMATA**, or it may be called by a single name of **myelomata**, are a form of blastomatoid process characterized by the formation of multiple tumours in the **bone-marrow**, associated with other lesions of the nature of general blood infection, such as the appearance of Bence-Jones albumose in the urine, progressive asthenia of vascular and muscular tissues, and in many cases with enlargement of the spleen.

Multiple or Diffuse Myelomata.

The condition appears to be of the nature of osteomalacia associated with the formation of greyish or reddish new growths in the skeleton, principally in the red marrow of the ribs, sternum, vertebræ and cranium, all arising usually simultaneously. It is also described as **Myelopathic albumosuria**. The marrow of the vertebræ, sternum, ribs, and rarely of the long bones are turned into growths of myelomatous nature. Sometimes, the skull and pelvis are affected as well. Thus it may be noted, that the lesion usually appears in other than long bones, in contradistinction to the giant-celled tumours. Myelomata attacks the flat, or irregular or membranous bones. The tumours are multiple and diffuse, and the area of the affected bones under X-rays, appears as if pierced by small foci, the spot of destruction not being confluent. Many small foci do not expand or destroy the cortex. Cysts may develop in many of them. Bence-Jones albumose are found in the urine in more than fifty per cent. of cases, which confirms the actual diagnosis. This albumose is suggested by some observers to be derived from the elastic tissue of bone. But such a condition doubtless proves the presence of severe nephritis. Due to the weakening of the bone produced by these masses pathological fractures of spontaneous nature occur frequently in these cases but they unite without giving much trouble.

**MACROSCOPICALLY** the masses of the growths are small, soft in consistency, reddish grey in colour, and sometimes hæmorrhagic. Each focus may confine itself without piercing the cortex, and some of those which perforate the periosteum may invade the neighbouring soft parts. These small tumours weaken the bone and the absorption caused by them may result in producing curvatures of the vertebral column.

**MICROSCOPICALLY** the pictures described by different observers demonstrate that probably more than one type of Myeloma may occur. One point is prominently pointed out unanimously and that is, the osteo-

clast type of giant-cells characteristic of giant-celled blastomatoid growth are never found in Myeloma. The growth may in some cases be composed of myeloblasts, or in others of plasma cells, round cells resembling lymphocytes, and cells arising from myelocytes or large enough to resemble myelocytes. The nature and origin of the cells are often uncertain, and opinions differ in their description; but it is probable that the bone-marrow cells of various types are the parent cells. The cells are separated by very little stroma which consists simply of a delicate meshwork of fibrous tissue in which they are loosely packed.

CLINICALLY the most characteristic phenomenon observed in more than 50 per cent. of cases is the appearance of Bence-Jones albumose in the urine. The test of the presence of this albumose can be very easily made by simply heating the urine. At a comparatively low temperature, usually at 50° C., the albumose is precipitated which starts to clear at 80° C., and is redissolved on boiling; but reappears on cooling.

Blood picture reveals a profound anæmia of progressive nature, otherwise it seldom shows any marked change, but myelocytes and plasma cells are detected in many cases. The progressive anæmia finally terminates the scene in death. We shall soon describe the relationship of this marrow-type of blastomatoid process with other primary blood diseases and describe how Myelomatosis constitutes a link with the blood diseases of primary type and some ill-defined malignant blastomata. We may from now remember that in the various forms of *leukæmia*, it is the marrow cells which form blastomatoid masses in the viscera in addition to those circulating in the blood. The latter feature throws into shade the overgrowths of the bone-marrow that may be present in splenomedullary leukæmia and lymphatic leukæmia by the constitutional symptoms produced in the circulating blood. In Myelomatosis on the other hand as well as in Chloroma the appearance of the blastomatoid nodes in

Myelomatosis is a link.

connection with the bones is the chief feature, whereas blood changes in them are absent, or anomalous, irregular and of little help for diagnostic purpose.

Local signs and symptoms vary according to the region affected, and the reader is referred to regional part of the Surgery for their details.

**TREATMENT** consists of more or less general therapy such as administration of arsenical compounds by injection, radium therapy, X-rays, etc.

Chloroma.

**CHLOROMA** is a blastomatoid process closely allied to Myeloma. The growths like Myelomata appear as periosteal tumour in the skull, vertebræ, ribs, and sternum; but its characteristic features are: (i) examined in the fresh state, a recent specimen presents a greenish colour which rapidly fades on exposure to the air; (ii) it presents a blood picture of an acute leukæmia. The colour may not appear in all cases, and the blood picture too is not constant in type or number of the leucocytes.

The growths are of the nature of infiltration of the periosteum, the infiltrating cells being fairly large rounded cells of the lymphoid type, lying packed in a delicate reticulum of connective tissue. The pigment has not been isolated and its chemistry is unknown.

It is a very rare tumour, and it usually affects the young people.

## (6) BLASTOMATOIDS IN CONNECTION WITH BLOOD CELLS.

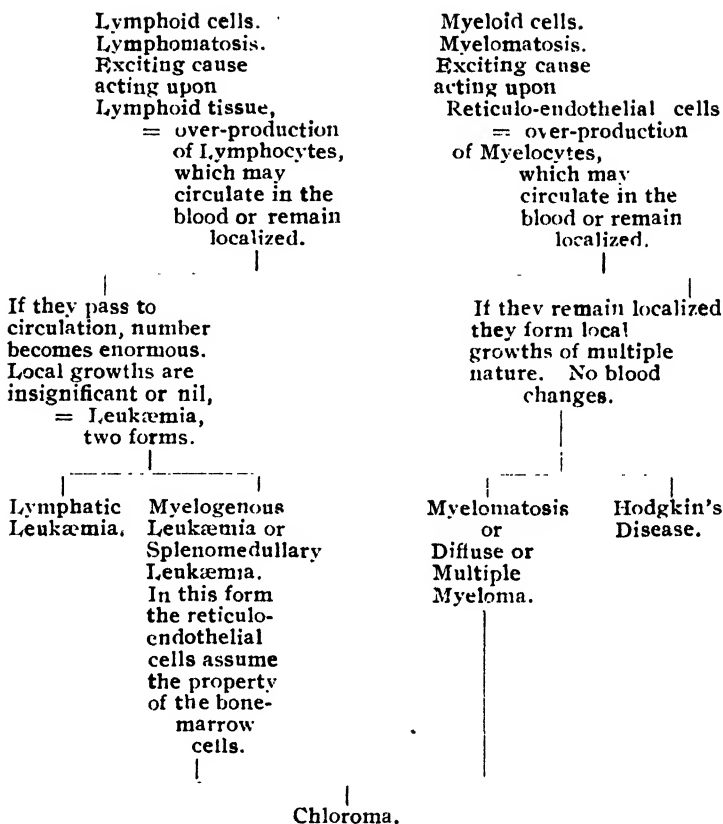
(6) Blastomatooids in connection with blood cells.

Blood is a liquid connective tissue and to mention it in connection with growths which are of course solid should excite in us more than usual interest. We find Myeloma confronts us with a blastomatoid condition which constitutes a *link* between the primary blood diseases and certain ill-defined Blastomata.

For instance at one side of the picture, *e.g.*, the lesions manifested in the blood, such as Splenomedullary

Leukæmia and Lymphatic Leukæmia, are not included under blastomata, because in all these pathological conditions the changes in the blood are their chief features, although the origin of the lesion actually takes place at the bone marrow and other allied tissues. Whereas Myelomatosis and Chloroma are conditions which exhibit distinct blastomic features in which the blood changes are anomalous and irregular, or absent, and the formation of neoplastic growths like nodes, in connection with bones is their chief feature. From such process again, round-celled sarcoma and lympho-sarcoma are but steps leading us into the clear region of blastomata.

Leukæmia as we know is a disease affecting the reticulo-endothelial system, the chief organs concerned being the lymphatic glands, the spleen, and the bone-marrow. According to the exciting causes acting upon the lymphoid or myeloid tissue the lesions exhibited are as follows:—



The above scheme will clearly show what a close relationship exists between Lymphatic Leukæmia, Myelogenous (splenomedullary) Leukæmia, Myeloma, Chloroma, Hodgkin's disease and Lympho-sarcoma.

### (7) BLASTOMATOIDS IN CONNECTION WITH LYMPHOID TISSUE.

(7) Blastomatoid in connection with Lymphoid Tissue.

Properly speaking, under this group fall all blastomatoid processes in connection with lesions in glands or Adenoid tissues. In considering the study of the surgery of the lymph glands it should be remembered that the

glands are made of two **different** tissues, *viz.*; (1) the **Lymphoid** cells which are contained in the vestibules or passages of the reticulum, (2) a **Reticulo-Endothelial network** composed of reticulum of fine fibrils in connection with which we come across a peculiar type of **endothelial** cells.

Any pathological process may therefore affect all the three structures mentioned above, *viz.*, (a) the lymphocytes, (b) the endothelial cells, (c) the reticular fibres; and in most of the instances, the latter two co-operate each other and react together.

The different clinical blastomatoid conditions met with are:

(1) Hodgkin's Disease. *Vide* photo plate No. XXXIV.

(2) Von. Mickulicz Disease.

(3) Lymphadenitis of various other glands, or their groups, and local glandular organs.

The detailed description of these lesions are included under the Disease of **Lymphatic** system and regional surgery, *vide* next volumes, as this is not the suitable place to describe them in detail.

### THE NEOPLASTIC ORIGIN OF LEUKÆMIAS.

At the present moment the theory of *neoplastic origin* of the leukæmias is engaging much attention and collecting a large following. The atypical cells of a malignant blastomic process endeavour to continue the functional characteristics of the typical normal cells from which they originate. One of the functional characteristics of all wandering cells of the blood is that when they extravasate out of the vessels in the tissues they *do not* excite any defensive reactive phase at the neighbourhood, or destroy the neighbouring tissues. Leukæmic cells in Leukæmias, *e.g.*, myeloses and lymphadenoses, behave in the same way. And when they infiltrate between the cells and tissues of any organ, *e.g.*, the liver, they do not excite a defensive reaction, and they do not destroy

The Neo-  
plastic  
origin of  
Leukæmias.

them although they may be neoplastic in their integrity of their birth. This explains the reasons why in leukæmias the infiltration found in the organs does not exhibit a destructive picture as is seen in other malignant tumours. Tumour cells derived from lymphoblasts and myeloblasts excite no reaction. According to Weber, this constitutes the most important argument in favour of the neoplastic theory.

(c) Blastomatooids of Muscular Tissue. Myomatosis.

### (C) BLASTOMATOID GROWTHS OF MUSCULAR TISSUE.

Blastomatoid growths in connection with smooth or involuntary muscle bear no surgical importance. The instances met with are as follow:—

(i) Overgrowths of muscle fibres of the coats of **arteries** and **veins** observed sometimes in fibromata are of blastomatoid nature.

(ii) Hyperplasia of the muscular tissue of the prostate observed in the **senile hypertrophy of the prostate** is apparently blastomatoid in nature.

(iii) **Adenomyoma of Uterus** possesses many characters of blastomatoid nature. They are still more interesting as regular gland tubules are found embedded in them.

(iv) Myomatosis has also been observed in connection with **subcutaneous** tumour of the leg. (Kettle).

(d) Blastomatooids of Nerve Tissue.

### (D) BLASTOMATOIDS IN RELATION WITH NERVE TISSUE.

Nerve tissues are very much liable to blastomatoid processes and for this reason it is sometimes very difficult to differentiate a genuine nerve tumour arising in connection with nerve fibres and nerve cells from a blastomatoid process. Tumours in connection with nervous elements both including the blastomatoid processes and blastomata are described clinically as Neuromata. But those com-

posed of newly formed medullated nerve fibres or ganglion cells are described as **true** Neuromata; and those which are blastomatoid processes arising from nerve sheaths and more or less constituted of fibrous tissue, histologically resembling a fibroma are described as **false** Neuromata. True Neuroma is very rare, and will be described under Blastoma.

Practically all neuromata commonly met with are false tumours. They are called *false* because it is not the actual nerve tissue that proliferates to form them when a tumour develops, but such growths take place in connection with the sheaths of the nerves, that is to say constituted of fibrous tissue. Three varieties of blastomatoid processes in connection with nerve tissue are met with, *viz.* :—

1. Localized Pseudo-Neuroma.
2. Diffused or Generalized Neuro-Fibromatosis.
3. Traumatic Neuroma, and Amputation Neuroma.

Three  
varieties.

## 1. LOCALIZED PSEUDO-NEUROMA.

The localized forms of Pseudo-Neuromata are composed mainly of fibrous elements. Some cases of this nature may take to malignancy, and malignant pseudo-neuroma can be detected by its being composed of sarcomatous tissues. Pseudo-Neuroma may grow from one side of the nerve, or the nerve fibres may spread around all over the growth; the latter being more frequent. The growth is freely movable; a condition which is clinically better elicited by attempting to move it from side to side, at right angles to the axis of the nerves, when it moves more freely, than when such an attempt is made along the course of the nerve in which the tumour develops. When these tumours arise from a large mixed nerve they are described as, (a) **trunk** Neuromata. Trunk neuromata are comparatively less painful than the subcutaneous variety described below; perhaps because these having less nerve fibrillæ and the growth being deeply situated. When they grow on a pure motor nerve no pain of radiating nature is

1. Localized Pseudo-Neuroma.

(a) Trunk Neuroma.

complained of, although they are sensitive. Complete paralysis or anaesthesia are never produced unless the growth turns malignant. In the cases where the tumour is deeply situated, the surrounding neighbouring structures may become invaded.

Trunk Neuromata grow more amongst women than men. They are generally found in the adults.

(b) The  
Sub-  
cutaneous  
Nodules.

(b) The next variety of localized form is the painful **subcutaneous nodules**. These grow in large number in connection with insignificant nameless small or fine superficial subcutaneous twigs. (Owing to their superficial nature these nodules are more exposed and are therefore very painful. When pressed or irritated, the tumour causes intense radiating pain. Exposure to cold produces a radiating neuralgic pain.

Treatment.

**TREATMENT.** If neuromata become painful producing symptoms of local or focal nature, they should be removed. The only point to remember in connection with such an operation is, to be careful of the actual nerve trunks from damaging them. If the continuity of the nerve is found to be involved in such a way as to make complete excision impossible, without damaging it, the portion of the involved nerve along with the growth should be removed, by dividing the nerve at the immediate proximal and distal aspects of the tumour. After removing the mass, the cut ends of the nerve should be brought nearer each other and united by sutures. Usually the sheath can possibly be divided on its long axis and the growth removed.

## 2. GENERALIZED OR DIFFUSE NEURO- FIBROMATA.

2. General-  
ized-Neuro-  
Fibromata.  
Or the  
Multiple  
Forms.

When many neuromatous tumours develop arising from the endoneurium of the primary nerve bundles, in very large numbers, they are described as **multiple or diffuse neuro-fibromata**. It is a condition of diffuse growths consisting of small elliptical or spherical shaped

nodules, the sizes varying from a small bead to that of an almond, or an egg ; which may sometimes attain a very large size. Sometimes the whole nerve trunk may exhibit uniform thickening or an enlargement of generalized nature, so much so that even a small twig may reach the size of a big nerve. The number of the individual tumours developing, may vary from only a few to a few hundreds in the same limb or at a particular area of the trunk of the individual ; most of which are hard in consistence, and are usually whitish in colour. They may or may not be encapsulated.

Diffuse Neuro-fibromata arise from the endoneurium, separating the nerve elements of the nerves, especially involving the whole nerve sheath. The tumours may develop at any part of the peripheral nervous system, including the sympathetics. But the most common sites affected, are the nerves of the cranium and the plexus of the trunk. The epineurium and perineurium are slightly thickened.

The growths may develop at any age of life. They grow very slowly. Sometimes several members of the same family are found to be attacked with the disease.

MICROSCOPICALLY,—the histology closely resembles that of a fibroma.

CLINICALLY,—the symptoms and signs are not very marked ; but when they develop in the cranial nerves or at the root of the spinal nerves, motor symptoms may become evident. In the orbit, exophthalmos may be produced. The tumours are sensitive to pressure, and these of them which are exposed on the surface may be very tender. Otherwise, the symptoms are of insignificant nature.

Any of the above varieties may turn malignant and become sarcomatous.

SURGICAL TREATMENT, is of little avail ; except trying to eradicate some of the bigger ones, and attempting to improve general health, nothing of much importance can be done.

Signs and  
Symptoms.

**Clinical  
Modifica-  
tions.**

Diffuse types of neurofibromata exhibit some different **clinical modifications**; viz. :—

(a) The Hard Fibro-matous type, which is practically described above.

(b) The Soft Plexiform type, or Myxofibromatous Neuroma.

(c) The Molluscum Fibrosum, including Pachydermatocele.

(d) Recklinghausen's Disease, or Multiple Neurofibromatosis with pigmentation of the cutis.

(e) The Elephantiasis type of Neuroma.

**(a) The  
Hard  
Diffuse  
Neuro-  
fibromata.**

(a) **THE HARD MULTIPLE TYPE.**—This is the typical variety of multiple neuroma described above. The other types described below are mere modifications of this type, and one may co-exist in association with any of the other varieties.

**(b) The  
Plexiform  
Neuroma.**

(b) **THE PLEXIFORM NEUROMA**, or the Myxofibromatous Neuroma is a modification of the diffuse type which occurs amongst very young people, or may be congenital. The special feature of this type is the softness and gelatinous nature of the tumours. Their consistence is soft giving a feel of a plexus of thickened tortuous and wormlike strands on palpation. These growths are confined to the distribution of a particular plexus of nerves; some of which may manifest the nature of the first type of the generalized neuro-fibromata. Plexiform Neuroma usually occurs in the nerves of the head and neck, rarely in those of the limbs. Very rarely sympathetic system may be affected.

**Clinical  
Features.**

**CLINICALLY**, the condition is manifested by many ill-defined swellings, consisting of a number of tortuous convoluted cord-like structures lying under the skin, on the line of the nerves; or on the area of distribution of anastomosing or contiguous nerves; which can be felt freely movable on one another lying in a loose tissue. These growths are rarely painful and seldom tender to touch, although the plexiform type is almost always sub-cutaneous. The commonest sites they develop, as stated





VON RECKLINGHAUSEN'S DISEASE.

above are the head and neck in the distribution of the trigeminal or superficial and upper cervical nerves. When removed, the growth appears like a mass or so many bunches of entwined strings of fine pearls.

This type rarely turns malignant and the prognosis therefore is rather better than in the former condition.

**TREATMENT.**—Big ones, may be excised for the sake of cosmetic purpose, and the result of excision is satisfactory; although it is not possible to remove them completely.

(c) **THE MOLLUSCUM FIBROSUM.**—Molluscum-fibrosus arises from the skin and develops in association with the terminal filaments of the cutaneous nerves. In this condition, the blastomatoid processes grow in large pendulous masses of fibrous tissues hanging from the surface of the skin. Molluscum-fibrosus is extra-vascular and softer than ordinary fibromata. In some cases, they attain enormous size, sometimes hanging in large masses of lobulated and folded overgrowths of the surface of the body. These growths are often associated with, and are closely related to, the Plexiform Neuromata, and von Recklinghausen's disease. *Vide* page 18.

(c) The  
Molluscum  
Fibrosus.

No treatment is of any avail.

Sometimes, the growths become so excessive that they resemble elephantiasis neuromata in the limb, described below. Such a condition of the molluscum is called **pachydermatocele**, which grows as an irregular hyperplasia, sometimes, especially when from the scalp, causing hideous deformity.

Pachyder-  
matocele.

(d) **RECKLINGHAUSEN'S DISEASE.**—This condition is a modification of multiple subcutaneous neuromatosis. The whole body including the trunk, limbs, head and neck is covered with soft almond or pea like projecting tumours some of which may attain a size as big as hen's egg. Sometimes hundreds of such small tumours may appear on the same person (*vide* photo plate No. XXXV) scattered all over the body. The palms of the hands and soles of the feet are often excepted. The

(d) Reck-  
linghausen's  
Disease.

only point of difference from the molluscos variety is its association with pigmentation of the skin. But this feature may not always be evident. In the early stage, the overlying skin remains freely movable over the nodules; which, in the later condition, becomes fixed and smooth, and with the increase of the growth in size becomes pedunculated.

No treatment is of any avail.

(e) The  
Elephan-  
tiasis  
Neuromata.

(e) **THE ELEPHANTIASIS NEUROMATA.**—This is a condition which outwardly resembles the features of inflammatory elephantiasis caused by *Filaria sanguinis hominis nocturna*, the latter so commonly met with in Bengal. This elephantiasis generally affects the legs and is caused by the extension of neuro-fibromatosis to the skin and cellular tissue of the limb. The affected extremity is swollen as a whole.

It begins in early life and is associated with diffuse general neuro-fibromatosis.

**TREATMENT.**—When the limb becomes too bulky, amputation is the only method to afford relief.

### (3) TRAUMATIC NEUROMA.

3. Trau-  
matic  
Neuroma.

After amputation or division of a nerve by trauma, with the healing of the wound, the cut end of the proximal part sometimes develops into a mass of neuro-fibro-cicatrictal tissue, by the prolongation of the nerve fibres. This happens by the continued regenerative process of the newly formed axis cylinders. The mass consists of such proliferated axis cylinders embedded in a large quantity of fibro-cicatrictal tissue.

Complete  
Division.

Traumatic Neuroma may develop after complete or incomplete division of the nerve. Such division may occur in civil practice, by cuts or injury caused by sharp fragments of glass, or machinery, or knives. In the battle fields, complete or incomplete division of nerves may be caused by missiles. If the nerve is completely divided, a **terminal neuroma** may develop. No sooner any nerve is cut, the two cut ends at once retract. The space

(i) Ter-  
minal  
Neuroma.

left between them is immediately filled with blood which is ultimately absorbed and replaced by granulation tissue. A bulb-like mass gradually develops by the proliferation of the fibro-cicatrical tissues, embedded in which, spaces are formed, which are filled with fine nerve fibrillæ coiled up in loops.

After an amputation most of the divided nerves develop Terminal Neuroma, which can be elicited by palpation of a bulbous mass. In some nerves, divided by accidental trauma, if the divided ends are provided with a scaffold of fibrous or other tissues, which can be used by the nerve cells as a bridge between the two cut ends, the proximal filaments may use such a scaffold to cross over the gap and re-establish the continuity.

Incomplete division may result by injury from glass fragments or fine cutting instruments in civil life, or by small conical bullets or fragments of high explosives in the fields. If the nerve is simply notched a gap is produced, which remains as such, by the traction of the divided fibres which stands in the way of reunion, and the embryonic axis cylinders may then proliferate and form a **lateral neuroma**. Such a neuroma may become adherent with the scar and reach the surface. In certain unusual circumstances, the nerve fibres at the centre of a nerve may be divided by the penetration of very small fragments of the missiles, when a **central neuroma** may develop in the same way as described.

Incomplete Division.

(ii) Lateral Neuroma.

(iii) Central Neuroma.

Traumatic Neuromata may give rise to a series of signs and symptoms ; e.g., irritative, trophic, atrophic, paralytic, etc., that is to say, all motor, sensory, trophic, and vasomotor signs and symptoms may develop. For the details of which and its treatment, *vide* Affections of Nerves.

Signs and Symptoms.

## GLIOMA.

Our conception of what constitutes a Glioma is now materially changing. A history of some kind of trauma

Glioma.

Glioma-  
tosis.

may be obtained from the majority of the cases, and it is therefore believed that the process is a mere reactive hyperplastic proliferation of progressive blastomatoid nature, rather than a blastoma. At least many of them are really **Gliomatosis** of progressive type, there remains to have no doubt. For the details the reader is referred to the next chapter.

### B. Blastomatoids from **metabolic** and **toxic** causes.

Blastomatoids arising from metabolic or **toxic** causes are usually found in the glands, *e.g.*, thyroid, *vide* photo plate No. XXXVI, prostate, breast, etc. Their ætiology has, to a certain extent, been described. The pathological changes will be described in the regional part of the Surgery.

## SUMMARY.

What is a Swelling? Swelling means a Tumour? But what is a Tumour? Clinically, Tumour means nothing but a swelling. Pathologically, it signifies a new growth: But Hypertrophy is also a new growth. All are cell-proliferations. Pathology of progressive overgrowths. Hypertrophy. Essential factors to produce Hypertrophy. Different causes or conditions where Hypertrophy is excited.

I. Hypertrophy, where the cause is physiological and the effect histologically normal, *viz.*,

- (1) Internal Secretion.
- (2) Inactivity.
- (3) Increased blood supply.
- (4) Hormonic disturbance.
- (5) Faulty embryonic arrangement.

II. Physiological hypertrophy from pathological causes. *Viz.*,

- (1) Septic inflammation.
- (2) Irritation of foreign body.
- (3) Infiltration due to overproduction.
- (4) Excessive work compensatory to strain.
- (5) Work compensatory to static action.
- (6) Work compensatory to analogous function.
- (7) Irritation of poisons.
- (8) Only excessive blood supply.
- (9) Neurotic disturbances.
- (10) May be adaptive.



PARENCHYMATOUS \* GOITRE.



III. Pathological causes producing pathological Hypertrophy. Hypertrophy here involves fibrous tissues only. *Viz.*,

(1) Fibrosis.

(2) Cirrhosis.

BLASTOMATOID GROWTHS. TABLE. The co-relations.

A. IRRITATIVE TYPES :—

Chemical. Parasitical. Infective.

(a) EPITHELIAL :—

(1) Surface Type.

(2) Glandular Type.

Cyst-adenomata. Molluscum Contagiosum. Blastomatoids of Dental epithelium.

(b) CONNECTIVE TISSUES :—

(1) Areolar Tissue.

Scrotal Tumours. Keloid. Molluscum Fibrosum. Fibromatosis.

(2) Adipose Tissue.

Diffuse Lipoma in Men.

Doubled chin. Peri-renal Lipoma. Adiposis Dolorosa or Dercum's Disease. Clinical Feature. Treatment.

(3) Blastomatoids of Cartilaginous Tissue.

(4) Blastomatoids of Osteal Tissue.

Subungual Exostosis. Dental Exostosis. Myeloma or Myeloid. Sarcoma or Giant-Cellled Tumour. Benign Giant-Cellled Tumours. Xanthoma Cells. Pathological Types.

(i) Periosteal Type.

Epulis.

(ii) Medullary Type at the ends of the long bones remote from the directions of the nutrient vessels. Clinical features. Sites. Very common at the ends of the bones which ossify last. Also at the jaw, tendon sheaths, bursa, and capsule of the joints. Signs, symptoms. Swelling of progressive nature. No redness or heat. Pain. Fracture. Deformity. Disability. Muscular Atrophy. Treatment.

(5) Blastomatoids in connection with Bone Marrow.

Multiple or Diffuse Myelomata. Myelomatosis is a Link. Chloroma.

(6) Blastomatoids in connection with Blood cells.

(7) Blastomatoid in connection with Lymphoid tissue.

The Neoplastic origin of Leukæmias.

(c) BLASTOMATOIDS OF MUSCULAR TISSUE. MYOMATOSIS.

(d) BLASTOMATOIDS OF NERVE TISSUE.

Neuromata: True. False. Three Varieties.

(1) Localized Pseudo-Neuroma.

(a) Trunk Neuroma.

(b) The Subcutaneous Nodules.

Treatment.

(2) Generalized Neuro-Fibromata. Or the Multiple Forms.

Signs and Symptoms. Clinical Modifications.

(a) The Hard Diffuse Neuro-fibromata.

(b) The Plexiform Neuroma.

Clinical Features.

(c) The Molluscum Fibrosum.

Pachydermatocele.

(d) Recklinghausen's Disease.

(e) The Elephantiasis Neuromata.

(3) Traumatic Neuroma.

Complete Division.

(i) Terminal Neuroma

Incomplete Division.

(ii) Lateral Neuroma.

(iii) Central Neuroma.

Signs and Symptoms.

Glioma.

B. BLASTOMATOIDS FROM METABOLIC AND TOXIC CAUSES.

## CHAPTER II.

### BLASTOMA AND TERATOMA OR EMBRYOMA.

In Hypertrophy, we found that the new growth is so consistent with its proportionate shape, as a mere enlargement of the original form of the organ hypertrophied, it is so consistent in its mode of growth carried out by our well-known physiological processes of nutrition, and it is so consistent in serving a definite purpose, ceasing in its progress of growth no sooner that purpose, for which, or the cause, or the requirement, which irritated it to grow, ceased to exist, that we never call a Hypertrophy a Tumour. But Hypertrophy is a **new formation** of tissues without doubt. Acromegaly, Gigantism are all exaggerated growths also. New growths like Sarcoma and Carcinoma will grow like a parasite at the expense of our body, inconsistent with its shape, inconsistent of the histological structure of the tissue which is different from the matrix tissue, inconsistent with the manner of its growth, inconsistent with any particular ætiology, rhyme or reason, never stopping or ceasing to develop; and these actions being continued haphazardly, automatically and autonomously. These latter ones, form a group of their own. They vary in **size**, they simulate or copy no known or existing physiological structure, organ, or limb; they vary in **form**; and they are distinctly threatening to our life, they grow or persist without subserving any physiological function and without any typical termination.

General Remarks. The consistency of growth and the utility to serve some purpose in the working of the system are the features in Hypertrophy.

We can therefore little use the term 'Tumour' to signify these latter deadly '**growths**'; and for the similar reason the term '**new growth**' cannot be used so generally for them without some special significance.

Neo-plasm  
means  
'New ele-  
mentary  
life'.

We, therefore, call these deadly group of growths as Neoplasm. Neoplasm means **new elementary** life; but by this term let us grasp and understand a special denotation and connotation of this word for growths, or swellings, which has nothing to do with the loosely applied term Tumour. We cannot use a term in any sense **clinically**, which is not accepted or corroborated **pathologically**. The nomenclature of **clinical terms** originates from general observations which is applied to particular cases. The logic here is, from general to particular, or more or less empirical; whereas, pathology will not accept a term unless that term is justified by scientific observation, reasoning, and proof, in each particular case, which is finally used in general sense. The logic in pathology is, from particular to general, which is a surer and more correct way of understanding a science.

We can, therefore, use the term Tumour as a common and general word exactly carrying the same denotation and connotation as 'swelling' or 'growth'. All Neoplasms therefore may be described off hand as Tumours but all Tumours are not Neoplasms. But no science will allow that sort of a vague designation.

Is Neo-  
plasm a  
living  
thing?

Although we designate these alien growths as 'new elementary life', we do not pretend to say this way or the other way that a Neoplasm possesses a life separate from ours, or independent of ours, while growing in our tissues, sucking our blood like a leech or growing like a parasite as a round worm. It may be that a certain kind of germ or life unit or protoplasm with life, which we would be able to detect later on, by some special microscope and some ultra-magnification, generate, multiply, and grow, at our tissue expense, and we do not pretend to say that such germs or units do not exist in these growths. In that way it would work as infection. If we accept that the activities of a cellular life are represented by growth, reproduction, and death, and if we take these signs as cardinal signs of life, we must

accept that a Neoplasm is a living growth which grows, multiplies, or reproduces and dies, and therefore it really consists of life, although it may be elementary. In the application of the term Neoplasm, again we encounter difficulties. We find, there are two distinct modes of origin of the Neoplasms. *Viz.* :—

(a) A tumour arising from Unipotential cells, or in other words cells capable of producing or forming only **one** variety of tissues. These are better described as **blastoma**.

(b) A tumour arising from Totipotential cell or cells, that is to say giving rise to **all** kinds of tissues of the body better described as **teratoma** or **embryoma**.

We find that Blastoma and Teratoma are holding a position far advanced in cell-proliferation from an ineffective swelling or growth as well as a Hypertrophy.

Neoplasm is occupying a position far away from Hypertrophy.

Our idea or knowledge of the genesis of these Blastomata and Teratomata is yet so dark, that we have not as yet been able to find out the exact ætiology of their origin. They are so peculiar in their own ways,

Our idea of genesis of Neoplasm is yet dark.

*e.g.* :—

(i) In **size**, some are as small as a small pea, some may grow enormously big.

Their ways of growth is peculiar, *e.g.* :—

(ii) In **shape** or **form**,—they may be flat, round, oval, spherical, spindle-shaped, or sigmoid or may look like a cylinder; and so, taking no definite shape of any particular organ or part of the body.

(i) In size.  
(ii) In shape and form.

(iii) The **surface**, may be smooth, or glistening, rough, or ulcerated, tubercle-like, or tubercous, raised, irregular, or of uncertain contour.

(iii) The surface.

(iv) In **consistence**, some are cystic, soft, or boggy, some are semi-solid, or hard, or dense, some yield a crackling like egg-shells, some are as solid, or as hard as ivory. Some are cellular, others are fibrous, and so forth.

(iv) Consistence.

(v) Colour.

(v) In **colour**, it may give a normal complexion or become pigmented or may take any shade.

(vi) Structure.

(vi) In **structure**, some may resemble only **elementary cells**, some may simulate a **particular tissue**, and some again may imitate an **organ**. The mimicing of a particular cell or tissue or organ may be so near completion, which is of course never perfect, that we classify our Neoplasms according to, (a) **cells** making up the main bulk of the growth and call them cell tumours or **cytomata**, (b) distinct **tissues** which make up the main bulk of the growth, but not in the least disposed to form any organ as we understand by such organ in physiology and call them Tissue Tumours or **histiomata**; (c) **organs** or parts of organs represented and situated in a curious position; *e.g.*, tooth or hair inside the ovary, and call them Organ Tumours or Organomata or **teratoma** or **embryoma**.

(vii) The progress of growth.

(vii) The **progress** of growth varies a good deal. Some grow very rapidly, others may take a very slow course, occupying practically the whole lifetime of a patient. The rapidly growing ones, may end their career causing death of the host in even a few weeks only. The slow ones may remain hanging in our body, without doing any harm excepting causing mechanical trouble by their size, weight, or pressure. The former ones are therefore called the **malignant Neoplasms**, the latter ones are called **benign Neoplasms**.

Malignant and Benign.

(viii) Their mode of invasion.

(viii) In their **mode of invasion** or occupation of areas, some are distinctly marked off from the surrounding neighbouring tissues, others keep no such boundary and these infiltrate and penetrate or **permeate** and grow anywhere and everywhere. Such growths may take place at the original seat of occupation, or it may take place at any other distant region, forming colonies, being transported by lymph or blood circulation from the original centre. There, they reproduce their like as secondary Neoplasms. These again may be the source of tertiary growths and so forth, they may multiply themselves like

pyæmic foci. These are called **metastatic growths**, and this process of **dissemination** is called **metastasis**. Such secondary growths are malignant in nature.

(ix) Their **history** is also very characteristic. (ix) Their History.  
Some may grow on progressively, like a hypertrophy. Such process may be slow or rapid. Others may abruptly take a **retrogressive** step and end in necrosis or gangrene, or end in necrobiosis such as atrophy. Or degenerations may follow, that is to say, hyaline, mucinoid, fatty, calcareous, or similar changes may take place. Necrobiotic changes generally occur at the oldest part of the growth.

In the progress of their growth, they cannot be always differentiated as **benign** and **malignant**. There is no exact line of demarcation between the two types, by which we can sharply divide them into two distinct classes of Neoplasms. By the term **benign** we do not mean to say that those growths are in any way friendly or kind to us in the same sense as we use the term in the clause "our benign government"; on the contrary the term 'benign' in pathology, is a qualification or adjective, which means accommodating oneself without much *immediate* inconvenience being produced to the host, but having its *own* interest subserved in full. Such growths will not interfere with the internal affairs of the host so long as they get their necessities for their existence in full. They do not penetrate into the tissues so long as their supply for their nutrition comes in through well organized and well **formed vessels and channels** of communication. They remain ramparted in a fixed wall. But they will go on growing, without concerning themselves with the mechanical and physical inconveniences that may be produced by such **harmless(?) expansion** of their colony. If we can extirpate them or excise them out **completely**, they won't trouble the host any further. They do not attempt building secondary colonies. The growth of the tumour and the

History of a Benign Neoplasm and characters.  
  
They grow by Expansion.

increase of substance is **uniform** and tends to be so throughout the whole mass in the majority of the cases.

History  
of a  
Malignant  
Growth.

They  
grow by  
permea-  
tion and  
Infiltra-  
tion.

The side of the picture of a Malignant growth is quite different. It is dark as ever. These growths will not remain inside a rampart or a wall, but will **infiltrate** and penetrate and **permeate** into the remotest tissues. They won't wait for well-formed vessels or channels of supply, but demand an abundant supply brought as hurriedly as possible by **any** means; as the growth of these tumours is independent of the physical condition of the host. They will expand, grow, penetrate, permeate, conquer, and assert themselves root and branch, and exploit as quickly as possible; and above all, the most dangerous feature being their sending millions of groups of their own kind to different regions to establish hundreds of secondary colonies, each developing into a well-established growth of its kind, which would behave exactly as the original focus. "Supply food", "supply necessities" are their constant cry. They will not wait for the host to carry their food to their own camp or abode, but enter into the farms and fields, workshops, and kitchens; *e.g.*, the bone-marrow which is a nursery where cells are made, to demand for their wants on **the spot**; and even then destroy and destruct the machinery. Very soon the host is exhausted, and annihilated; the tumour itself making the first claim upon such resources as remain to be drawn upon, right up to the last, all throughout its career Extirpation or excision of such a colony is **impossible**. The secondary colonies will each grow out as big and as **malignant** as the original one.

But benign growths do not always remain benign. Some of them may turn malignant at any phase of their career, and some would form secondary colonies.

For the above reasons it is better to classify them as Non-Malignant and Malignant, instead of Benign and Malignant.

## GENERAL STRUCTURE OF NEOPLASMS.

A Neoplasm consists of two primary parts of tissues; *General structure of Neoplasms.* *Two primary parts.*  
*viz.*, (a) the Neoplastic **cells** proper or the **parenchyma**; (b) the connective tissue matrix or the **stroma**, that is to say the frame-work or skeleton. It must be understood that a cell is derived from a pre-existing parent cell of the body and resembles the parent cell more or less closely.

(a) The **parenchymatous** cells actually determine the nature of the Neoplasm. In malignant growths, they do not appear to be exactly like the well-developed histological cells, but seems to take to the embryonic kind. That is to say, they revert to the primitive or **atypical** type. In non-malignant tumours the resemblance to the parent tissue is well-marked and the cells are more or less **typical**. *(a) The Parenchyma or the proper Neoplastic cells.*

(b) The **frame work** or skeletal part of the neoplasm called the **stroma**, consists of the connective tissue of the structure in which the neoplasm started growing. This structure varies considerably in amount according to the nature of the neoplasm, the different parts of the same neoplasm, and the organ or structure where it grows; *e.g.*, in epithelial or glandular type of tumours the two constituents are easily distinguishable and the matrix tissue may be more abundant than the parenchymatous element, whereas in the tumours of connective tissues the reverse is the case in most of the instances. *(b) The connective tissue stroma or Matrix.*

The **blood vessels** develop in the frame-work of the matrix or stroma made of connective tissues. *The blood vessels.* These vessels nourish the 'growth'. They are well formed and supplied by the host as the result of reaction. They are usually well developed in the non-malignant type; as in this type, the stroma and the parenchyma grow side by side. In the malignant type on the other hand, they are represented only by some spaces, sinuses, or chinks in the parenchyma, or utmost, they are formed of thin-walled capillary channels as results of additions by annexations, or fresh formation. The reason is

obvious and is explained above in general terms. Their demand is quick and peremptory. Of the two primary functions of a cell, *viz.*, growth and work, their function is devoted more to growth or sometimes to *growth* only than to execute any *work*.

Nerves.

The **nerves** are not detected to grow inside a neoplasm. New growths are devoid of any sensation. In Non-Malignant growths the skin covering them retains its sensory function. This is not always the case in Malignant growths. A malignant growth may surround a nerve but none are found in the substance of the growth, those found are limited to the periphery.

Lymphatics.

The **lymphatics**, as we know, are provided in all tissues for nourishment, and *drainage* to remove the metabolic products, if a tissue is to live. It is obvious that a new growth will do the same if it is to live and grow. In a new growth the lymphatics consist of simple *lymph-spaces*, and their metabolic products are thrown into the general stream by these lymphatics, in which their parts are also carried away.

Function of the affected tissue.

In Non-malignant neoplasms, the **functions** of the tissues from which it is derived may be retained, which is generally the case. In Malignant growths, the functions of the tissues **are not** retained, which is also generally the case with the latter. Exceptions will be discussed later.

Terminology.

**TERMINOLOGY.**—We call the neoplasms according to the histological nature of the tissue of which they are principally composed. *Viz.* :—

From Epithelium it develops into Epithelioma.

From Endothelium it forms Endothelioma.

Similarly Fibroma develops from fibrous tissue, Lipoma from fat, Neuroma from nerves, Osteoma from bones, Myoma from muscles, and so forth.

The suffixes 'mas' (English plural) or 'mata' Latin plural signifying plural growths, are used without invidious distinction between them.

Compound terms such as Fibro-myoma, Fibro-chondroma (cartilage), Lymph-adenoma, etc., are used to signify neoplasms which show the complex nature of their formation in which both the tissues named keep an almost equal dominance in their development; or the term succeeding may be used to signify the comparative predominance of that tissue only, as we use our surname to signify the family of our birth.

We also signify the particular nature of the parenchyma by expressing the histological character of the **cells**, of which the neoplasm is principally composed; *e.g.*, as normal epithelial cells might be squamous, columnar, spheroidal, etc., if a neoplasm is composed of any such particular form of the above cells, we call it Columnar-celled Epithelioma, or Squamous Epithelioma, etc., as the case might be. Similarly the terms "giant-celled" (tumour), spindle-celled, round-celled, etc., are the adjectives used to signify their additional "virtues". All malignant epithelial tumours are grouped together under one name called **carcinomata**, and all malignant connective tissue tumours are classed together as **sarcomata**.

But, the above nomenclature is convenient for clinical description to help us in giving the additional information of their morbid histology. It does not offer us much scientific basis to **classify** our neoplasms.

The present Nomenclature does not help the classification.

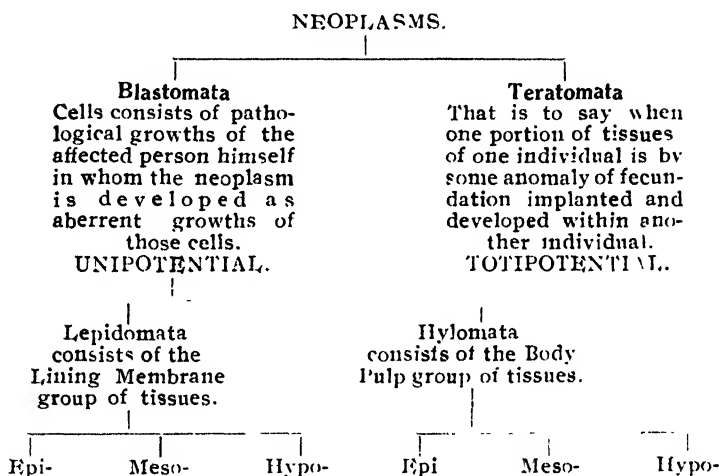
## CLASSIFICATION.

Moreover if we try to keep the consideration of both **histology** and **ætiology** together, in our mind, and in addition to the above factors, try to classify them according to the three **histogenic** or **embryonic** groups of tissues of their origin, such as the epiblast, the hypoblast, and the mesoblast; and if such classification is possible, the pathological designation would become more complete and may be to a certain degree scientifically perfect. Adami gives a scheme of this

Pathological Classifications.

Adami's  
Scheme.  
Modified.

kind. We should describe his classification in detail later. Here we add a short table to understand his views generally. Adami's main point is, that *each* of the three primitive layers (the trilaminar blastoderms) of the embryo consisting of the epiblast, the mesoblast and the hypoblast, gives rise to **two** orders or groups of constituting cells, *viz.* (a) those forming the **lining membranes**; and (b) those forming the **pulps** or solid tissues.



The term "Unipotential cells" means, cells capable of forming only **one** variety of tissue. "Totipotentia" means, cells capable of producing **all** kinds of tissues of which the human body is composed.

Powell-  
White's  
classification.

The above classification is simple and scientific and the nomenclature on such basis would give us the exact denotation and connotation of the terms applied giving us an idea from two different points of view, *viz.*:—(a) histological, (b) histogenic or embryological. But this classification though classical is not very convenient from practical clinical point of view. In that respect Powell-White's classification seems to be universally accepted, which is detailed below. We have

already stated that a Neoplasm may consist of three principal structures, *viz.*:—(1) Cell-forming (2) Tissue-forming; (3) Organ-forming. Accordingly they are described as **cytomata** or Cell Neoplasms, **histiomata** or Tissue Neoplasms, and **organomata** or Organ Neoplasms respectively. From this point of view it is better to use the terms **blastoma** and **teratoma** instead of Neoplasm, as the former would include all Cytomata and Histiomata in one word, and the latter will explain the features of all Organomata.

Blastoma is a better term.

But, before we describe such a classification on both histological and histogenic grounds in detail, it would help us to recapitulate the further course of development in the embryo after the formation of trilaminar blastoderm, which consists of the Epiblast, the Mesoblast and the Hypoblast. That is to say, it will help us in remembering the scientific method of classification as formulated by Adami, and also enable us to combine Adami's method with that of Powell-White, which we shall presently describe below for more practical convenience.

Embryonic Development.

## DEVELOPMENTS.

From each of the above embryonic layers as we may recollect from our Embryology, certain organs are formed as stated below in the following list:—

The three Embryonic Layers: Developments of.

### 1. FROM EPIBLAST.

1. Organs, etc., from Epiblast.

A. THE LINING OR PROTECTIVE EPITHELIUM OR LEPIDIC TISSUE:—

A. Lining or Protective Epithelium.

(i) The epidermis and its **appendages** which include the sweat, sebaceous and mammary glands. But it should be noted carefully that **dermis** or the true skin is not formed of this epiblastic layer. **True skin is a mesoblastic tissue**, and is therefore not included under

the **lining** tissue, but it is consisted in the **pulp** of the body.

(ii) The epithelium of the mouth, **adamant**, or **enamel of the teeth**; but not the cement or the crusta petrosa, and the pulp of the tooth. Both these structures are mesoblastic in origin.

(iii) The epithelium of the nasal passages.

(iv) The epithelium of the glands opening on the skin, and into the vestibule of the mouth, and nasal passages.

Body  
Pulp.

## B. THE BODY PULP EPITHELIUM, OR HYLIC TISSUE:—

(i) The muscular fibres of the sweat-glands. All other muscular fibres are mesodermic in origin.

(ii) Nervous system as a whole, both central and peripheral.

(iii) The epithelial structures of the sense organs.

2. From  
Meso-  
blast.

## 2. FROM MESOBLAST.

### A. THE BODY PULP OR HYLIC TISSUE:—

(i) All the muscles and muscular structures of the body.

(ii) The whole endo-skeleton.

(iii) All the connective tissues of the body.

(iv) The urinary and generative organs; *e.g.*, kidney, testicle, ovary, uterus and prostate. But it should be noted that the epithelial lining of the bladder and the urethra does not develop from the mesoblast. It is a pure **hypoblastic** tissue **not epiblastic**.

(v) The true skin.

B. Lining  
Membrane.

### B. THE LINING OR LEPIDIC TISSUE:—

(i) The vascular system including the lymphatics, the spleen, and the serous membranes. It is at this situation that a very important factor is involved, in connection with the **lining** of the interior of the whole

of vascular system, including the heart, arteries, capillaries, veins, lymphatics and the serous membranes, such as the pericardium, the peritoneum, etc. This **lining** is formed by **endothelium**. It is not epithelium, but a structure *similar* to it, and unlike the latter or those epithelium arising from the Hypoblast described as **mesothelium**, the endothelium is **mesoblastic in origin**. The importance of this epithelial-like structure arising from mesoblast has some direct bearing on the development of neoplasms which will be described later on. The special name **endothelium** should be used exclusively for the kind of tissues it represents and they should not be described by any other name but endothelium.

Endothe-  
lium.

### 3. FROM HYPOBLAST.

#### A. GLANDULAR OR SECRETING EPITHELIUM, LIPIDIC TISSUE:—

(i) The epithelium of the alimentary canal from the inner side of the teeth to the anus. Here again, it should be remembered, that up to the cardiac end of the œsophagus it is **squamous** in type, and the same type starts again at the lower third of the anal canal.

(ii) All the digestive glands which open into this major portion of the alimentary or digestive tract, including liver and pancreas; but not the mouth or the enamel of the teeth. These are epiblastic in origin.

(iii) The epithelium of the bladder and urethra. It should be remembered, that the lining of the pelvis, of the kidney, the ureter, the bladder, the vagina and cervix uteri are covered by **squamous** cells.

(iv) The epithelium of the respiratory tract.

(v) The epithelium of the Eustachian tube and tympanum.

(vi) The epithelium lining of the vesicles of the thyroid.

(vii) The epithelial nests of the thymus.

3 Organs,  
etc., from  
Hypoblast.  
A. Lining  
the glands  
and there-  
fore Secret-  
ing Epithe-  
lium.

B. Body  
Pulp.

What does  
Adami say?

B. PULP or HYLIC tissue:—The cartilages, notochord, the liver, the pancreas, etc.

Now, to follow Adami more easily, one can understand from the above list of tissues and organs developed from the three embryonic layers, that not only does the epiblast form the **lining**, or **covering**, or **protecting** epithelium of the body, or Adami's **lepidic** tissue and the structures immediately derived from it, such as the skin and its organs and appendages namely the sweat glands, the sebaceous glands, the mammary glands, etc., but an ingrowth of the same epithelium develops into solid or **pulp** structure such as the central nervous system; or according to Adami's terminology as the **hylic** tissue. And again the hypoblast not only gives rise to solid **pulp** or **hylic** tissue in the notochord and outgrowths from it; *e.g.*, the liver and pancreas; but it also constitutes the **lining** membrane of the respiratory and alimentary canals. So, also in the mesoblast, which is really composed of cells derived from both epiblastic and hypoblastic layers, a differentiation into lining tissue and pulp tissue is observed; *viz.*; (1) **mesothelium** or *lining* membrane of the primitive body cavity, and (2) **mesenchyme** or body *pulp*. From the mesothelium again are developed the lining membranes of the serous cavities (the endothelium), and organs such as kidney and suprarenals, and also solid outgrowths as striated muscles. From the mesenchyma are developed the lining membranes in the endothelium of blood and lymphatic vessels and spaces, and the connective tissue of the body.

Let us now examine Adami's scheme in greater detail. Let us see first how his scheme falls with the list above described with reference to histogenic or embryological development of **typical** cells and tissues. We shall first describe the scheme with reference to the **typical** cells and then proceed to compare it with their conditions of **atypical** transformation.

Table  
T.T.T. and  
A.C.T.



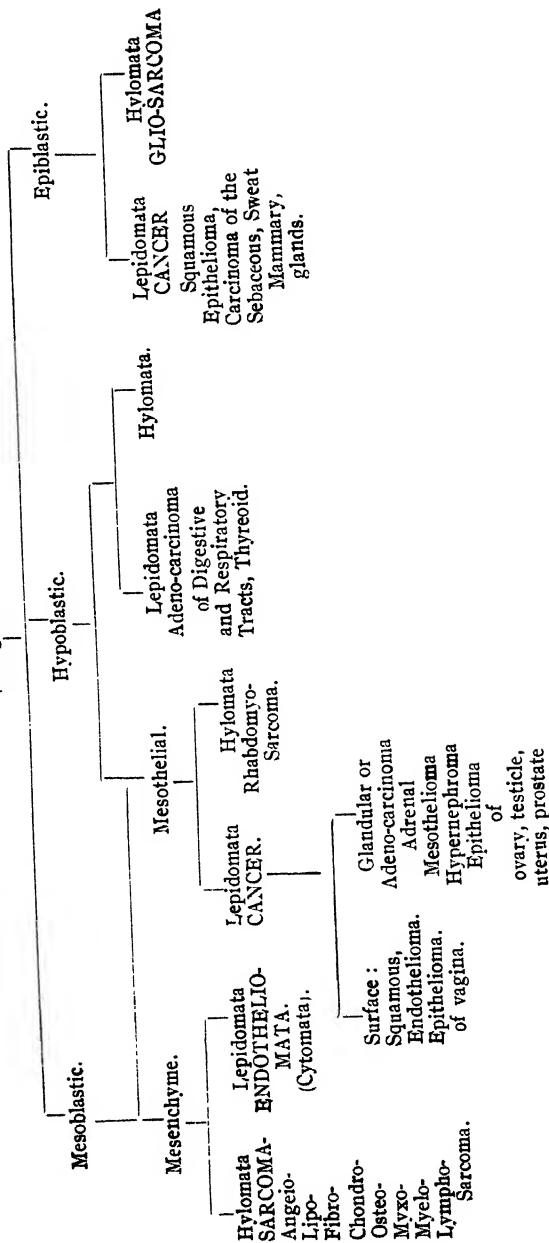
TABLE No. XX

Now when we recast the same Table with ATYPICAL cells let us see the result :—

A. C. T.

# ATYPICAL CELL TUMOUR

(Malignant)



The above complete description of the origin of tumours based on different histogenic tissues, is not only very helpful from point of view of classification, as it accommodates every type of tumour, but helps us to solve or attempts to solve the ætiology of the neoplasms. The following classification of neoplasms, tabled after Powell-Whites' formulas but modified and superadded according to embryonic origin, is a convenient clinical description of all types of neoplasms met with:—

## BLASTOMATA.

## A. HISTIOMATA OR TISSUE TUMOURS.

The neoplasms in this group manifest and exhibit distinct tissues, but they do not arrange in a physiological manner to **form organs**. These are:—

A. Histioma or Tissue Tumours.

## I. MESOBLASTIC.

I. Mesoblastic.

1. Endothelial Tissue.	{	ENDOTHELIOMATA. (Histiomata)		1. Endothelial Tissue.
		(1) Lymph-angioma.		
		(2) Hæmangioma.		
DESMOMATA.				
2. Supporting or Connective Tissue Tumours. Desmomata Lymphomata or Lymphoid Tissue Tumours.	{	(1) Lipoma = Fat.	2. Connective Tissue.	
		(2) Fibroma = Fibrous Tissue		
		(3) Chondroma = Cartilage.		
		(4) Chordoma = Notochord.		
		(5) Osteoma = Bone.		
		(6) Odontoma = Tooth.		
		(7) Myxoma = Mucous Tissue.		
		(8) Glioma = Neuroglia		
		(9) Lymphoma = Lymphadenoid Tissue.		
		(10) Myeloma = Bone-marrow.		
MYOMATA.				
3. Muscular Tissue.	{	(1) Leiomyoma.	3. Muscular Tissue.	
		(2) Rhabdomyoma.		

## II. EPIBLASTIC.

## NEUROMATA.

1. Nerve Tissue.	1. Nerve Tissue Neuromata.	{	(1) Myelinic Neuroma = Medullated Nerve.
		{	(2) Amyelinic Neuroma = Non-medullated Nerve.

## EPITHELIOMATA.

2. Epithe- lial Tissue.	2. Epithelial Tissue.	{	(1) Squamous.	{	(i) Hard and Soft Warts or Papilloma.
		{	(2) Columnar.	{	(i) Adenoma = GLAND.
		{		{	(ii) Papilloma = STALK.
		{	(3) Spheroidal.	{	(i) Adenoma = GLAND.
				{	(ii) Papilloma or Papillifero- us Cystic Adenoma.

B. Cyto-  
mata or  
Cell  
Tumours.

## B. CYTOMATA OR CELL TUMOURS.

These neoplasms are composed mainly of **atypical cells**, chiefly in the growing portion, but, they do not arrange themselves to form any particular tissue. These are :—

**I. EPIBLASTIC CELLS.**

1. EPIBLASTIC CELLS  
Carcinoma or Cancer  
proper by Epithelial  
Cells.

- (1) CARCINOMATA
- (i) Squamous { Malignant Warts  
or Squamous-  
celled  
Carcinoma
  - (ii) Spheroidal { Carcinoma-  
Simplex, or  
Carcinoma of  
Glandular  
Epithelium
  - (iii) Columnar { (i) Adeno-Car-  
cinoma  
(ii) Malignant  
Papillary  
Cyst-adenoma
- (2) NEUROCYTOMATA

I. Epi-  
blastic  
Cells.  
Epi-the-  
lial Cells.

**II. MESOBLASTIC CELLS.**

(1) Endothelial Cells.

- (1) ENDOTHELIOMATA.  
(Cytomata).  
Sometimes called Endo-  
thelial Carcinoma.

II. Meso-  
blastic  
Cells.

(1) Endo-  
thelial  
Cells.

(2) Connective Tissue  
Cells.

- (2) DESMOCYTOMATA.  
(SARCOMATA)
- Pure Sarcoma.
- (i) Round-Celled.
  - (ii) Spindle-Celled.
  - (iii) Giant-Celled.

(2) Connec-  
tive  
Tissue  
Cells.

(3) Lymphoid Cells.

- (3) LYMPHOCYTOMATA.

(3) Lym-  
phoid  
Cells.

(4) Muscle Cells.

- (4) MYOCYTOMATA.

(4) Muscle  
Cells.

(5) Mixture  
of In-  
different  
cells.

(5) Mixture of  
Indifferent Cells.

{ (5) BLASTOCYTOMATA.

III. Pig-  
mented  
Tumours.

### III. PIGMENTED TUMOURS.

IV. Hyper-  
nephro-  
mata.

### IV. HYPERNEPHROMATA.

V. Organo-  
mata.  
Teratoma.

### V. ORGANOMATA OR TERATOMATA OR EMBRYOMA.

In the neoplasms of this variety distinct organs or parts of organs are developed pathologically.

These are called Teratomata. Any intermixture or combination may of course take place and develop into compound types.

### CLINICAL CLASSIFICATION.

Clinical  
Classifica-  
tion and  
Patho-  
logical  
classifi-  
cation.

The above varieties practically exhaust the different pathological types of neoplasms that are clinically met with, so far as our present knowledge of neoplasm goes, which is of course still incomplete. It is fair to admit, and it would be dogmatic to assert that the question of Hypernephromata or Melanomata could yet be solved so far as its histology is concerned in Powell-White's classification.

The list as described after Adami is somewhat unwieldy and it is not always possible to practically diagnose many neoplasms which exhibit characteristic pleomorphism, *e.g.*, Hypernephromata, and classify them under their actual pathological heading unless we submit the tissue under microscope and closely examine them.

Clinical observation and classification does not at first depend on pathological classification, although the latter is of course the scientific method of classification. Pathological classification is logically correct. It proceeds from the examination of many particular instances, each being thoroughly scrutinized and credited of its merits or ex-

cluded by demerits, and then progresses from the less general to the more general. Whereas, on the other hand, clinical classification is based on deducing from the more general to the less general and is deductive in nature. We often proceed to describe something from superficial, vague, and sometimes incompletely observed facts, which may be confirmed and corroborated and found correct by pathological and other scientific investigations, or may be found to be wrong.

By our clinical observations as to its easy extirpation, its slow growth, its un-offending nature of attacking the host, or otherwise, we call a growth a **benign** or **non-malignant** or a **malignant** one. We have already explained that the term benign is not a happy term. One which is called 'benign' to-day, may start a dangerous exploitation and at anytime turn malignant; it is therefore often not easy to assign a particular growth to its class.

Surgeons use the terms Benign and Malignant on clinical observations.

Clinically we classify neoplasms as Non-Malignant and Malignant. The terms tumour and cancer mean nothing; *e.g.*, by the term cancer some may vaguely mean any form of **malignant tumour**. By our increasing knowledge in pathology now-a-days, the term **cancer** is restricted to denote **carcinoma** only, both clinically as well as pathologically. In our detailed description of tumours we shall describe them only under two broad headings, *viz.*, Non-malignant and Malignant, adding their morbid histology under each individual case. The broad features of these two groups are the following :—

Non-Malignant and Malignant.

Features.	Non-Malignant	Malignant.	The Broad Features of both.
FORMATION.	TYPICAL.	ATYPICAL.	
Resembling homologous normal histological structure of their cells and tissues.	Mostly or nearly complete.	Always incomplete	

Features.	Non-Malignant.	Malignant.
Age.	Younger people or, may be congenital.	Usually after adult age, but not necessarily
History of incidence.	Nil.	There is generally a history of irritation which may be infective, mechanical, thermal, chemical or otherwise.
Progress of growth.	Slow or stationary.	Rapid.
Power of continuous growth.	May be present	This is the most important attribute of malignant growth.
Capsule of fibrous tissue. This is the most important diagnostic point.	Generally always present.	Generally always absent.
Formation of the blood vessels.	More or less into complete vessels.	Usually thin walled and incomplete. Mere interspaces without, or with, little formation of vessel wall.
Embryonic or vegetative character of cell.	Little or no trace as a rule.	Anaplasia well marked. Generally embryonic or aberrant in type.
Presence of Mitotic figures in the nuclei of cells indicating rapid growth.	Nil.	Very marked.
Metastatic growth.	Nil.	Always prominent.
Infiltration.	No local infiltration.	Diffuse infiltration both local and focal.

Features.	Non-Malignant.	Malignant.
Anæmia. cachexia signs of general toxæmia, sepsis, infiltration of glands, etc.	Not present unless a malignant turn is taken.	These are really characteristic fea- tures of malignancy and is always pre- sent in Malignant neoplasms.
Recurrence after removal.	Rare ; if completely extirpated recur- rence is hardly possible.	Usually followed by recurrence, absence of which is very rare in cases of operation.
Ulceration.	Nil unless excited.	Prominent.
Necrosis or tendency to central degenerative changes.	Nil.	Prominent.
Hæmorrhage.	As a rule absent.	Prominent.
Inflammatory changes in the surrounding tissues.	Excites some reactionary fibrosis.	Usually found.
Relation to stroma.	A basement is usually present.	Absent.

The above differential points between the features of malignant and non-malignant tumours, are really based on pathological grounds. On strictly clinical grounds, any tumour which causes the death of the host by its effects on the host, direct or indirect may be described as malignant. From pathological point of view, a malignant growth, although invariably fatal is not called so on account of its quality of fatality, but a tumour is called malignant if it should possess the *features of malignancy* described in the above Table.

We shall however describe all the above features in greater details at their proper places subsequently.

**ÆTIOLOGY OF NEOPLASM.**

**Ætiology.** We are not yet definitely sure of the ætiology of Neoplasm, although sometime or other all the possible ætiological factors known to us (*Vide* Volume I. Part I. Chapter I.) have been brought to the fore front to account for its causation. Neoplasm varies in its incidence in different regions, amongst different animals, peoples, according to climates, degree of the advancement in civilization, different ages and periods of history, social status, age, sex, occupation, mode of life, food, and even to different tissues, organs, and anatomical regions, etc., in such divergent ways that no fixed clue could yet be found out which would definitely lead to any proved or universally accepted cause. Therefore there is no end of theories which are not hypotheticated to be responsible for its causation. The following are the most commonly accepted theories, *viz.* :—

**I. Virchow's theory of irritation.**

**I. VIRCHOW'S THEORY OF IRRITATION.**

Constant irritation is a factor which is accepted by all surgeons, as well as pathologists to be a productive source of new growths. Hypertrophy and hyperplasia are always caused by irritation, helped by excessive nutrition; but since in these conditions there is a typical termination, which is entirely absent in the case of Neoplasms the difference between the two conditions is obvious. The irritation may be of the following natures :—

**(i) Mechanical Irritation.**

**(i) MECHANICAL.**—In the warmer regions of India, cancer is not so common as in the colder upper countries of this peninsula. In India, irritation is found to be almost a constant factor in practically all cases of neoplasms; and this factor is so universal that it appears as if all cases of malignant neoplasms are caused by some kind of irritation. In Southern India extending from Orissa, constant irritation caused by the habit of incessant chewing of tobacco leaves with betel and *pan*,

is found to be a common cause of producing malignant growths in the oral cavity. Neither the *pan* leaves nor the betels chewed as they are done in Bengal, Behar, and U. P. produce cancer. Dried powdered tobacco leaves mixed with lime are commonly kept under the tongue by the people of upper India, but even raw tobacco used in that way does not seem to produce cancer in any marked degree, to stamp such an use of it as the causative factor. It is the mixture of the three ingredients mentioned above, accompanied with constant irritation of carious and rough teeth, which seems to induce the growth. The clay pipe cancer on the lips of working people in England, is due to irritation of constant thermal nature and mechanical pressure on a particular point produced by the use of rough clay pipes.

(ii) THERMAL.—In Kashmir the Kangri, a charcoal brazier used under the clothing for warmth, is a fruitful cause of producing constant thermal irritation. Women who are very fond of using them, even of comparatively younger age, become the subject of cancer of the skin. Engine drivers suffer from tumours on the skin of the same type as the result of long exposure to the heat of the fire. *Vide* photo plate No. XLII.

(ii) Thermal Irritation.

(iii) CHEMICAL.—Works necessitating constant handling and contact of soot and certain forms of tar, paraffin, pitch, petroleum, shale oil, often give rise to cancer; and probably chemical irritation of some particular chemical substance is the cause. Some observers describe this irritating substance in the tar and soot, as something of a special chemical nature which excites the development of cancerous neoplasms as **auxetic** substance. These observers state, that these auxetic substances stimulate cell division. Auxetic substances are present in soot and tarry materials, but not in other similar substances such as coal dust and blast furnace pitch; and the latter is not found to give rise to cancer. Very few cases of cancer are seen in the coal mining districts of Bengal. In England, the people are

(iii) Chemical Irritation.

strongly resenting the use of tar in tar-macadam roads on this ground, and the newspapers suggest, that the increase of cancer in England may be due to increased use of tar in road construction.

Not only the above, but quack doctoring over Non-malignant growths by means of the application of irritative chemicals, is a very constant factor of producing malignant ulcers. Such instances are often found done on elephantiasis of the scrotum. Adenoma in the rectum is sometimes turned malignant by such applications by so-called piles doctors practising in Bengal as 'Piles Specialists'. It has also been observed, that repeated administration of **arsenic** for psoriasis and other skin diseases has resulted in the development of cancer.

(iv) Infective Irritation that is Irritation plus broken surface or damaged tissues probably excited by some infection seem to be a fruitful cause.

(iv) INFECTIVE.—In India cancer is more commonly met with at the mouth, liver, penis, and rectum in the males, and cervix of the uterus, mamma, and the liver, in the females. In every one of these instances some kind of **irritation** associated with some **infection** is observed. Simple constant friction or mechanical irritation is never, or hardly ever, found to be followed by cancer. In that case every '*palki* bearer' would have got cancer on his shoulder. Unless the affected cells or tissues are previously damaged by infection, or the surface becomes raw or ulcerated by infection, or by chemical irritation, such hypertrophied growths such as '*palki* bearers' bursa, or as a matter of fact any non-malignant new formation, such as a scrotal tumour, does not show malignant development.

Syphilitic lesions of the penis, infective irritation in the uterus, amœbiasis of liver, chronic infection of mamma, chronic piles or tuberculous sinuses, ulcer and leukoplakia in the tongue, bilharzia infection of the genito-urinary tract, long standing lupus and tuberculous ulcers etc., are fruitful sources of pre-disposing cancer; if the lesion becomes subject to a constant irritation. The cells and tissues become vulnerable, being already in damaged conditions. These cases usually give

a history of 15 or 20 years before they exhibit malignant transformation.

Malignant **transformation** of ulcers and benign tumours is more common in the east than primary carcinomata. In the colder regions of India, such as in Nepal, the incidence of malignant cancer, as well as malignant transformation of ulcers and growths, are as common as in the west.

(v) **TRAUMATIC**.—Direct wound, fracture or blow is often a fruitful cause to excite malignant growths, *e.g.*, Sarcoma. It is possible that during the process of regeneration and repair, Nature's reparative attempts may exceed or even go beyond the limit in these cases, and proliferation of tissues continued, or the normal Tissue Tension, to be explained later on, is disorganized; the irritation of the trauma having acted as an exciting or a productive cause.

(v) Traumatic Irritation.

Implantation cysts or Dermoid acquired from injury may arise from similar causes of irritation.

(vi) **X-RAYS IRRITATION**.—X-rays irritation, or frequent exposures of tissues to such electric rays, produces typical cancer, on the parts actually exposed for a long time, usually more than five years. Hands up to the wrist and face down to the neck are the parts affected.

(vi) X-rays Irritation.

## II. PARASITIC THEORY.

**PARASITIC THEORIES**.—A non-malignant growth may show malignant transformation, but such transformation, is not a peculiar feature of those tumours alone. An ulcer or a hypertrophy may show the same inclination towards malignant degeneration. Such a phase is an accident, or an additional change of a mass of tissues, or growth, or an ulcer acted upon by some additional *factor*. This factor may be an additional contamination by some other secondary agent acting on such a growth or swelling, such as parasites or germs, or ultra-bacteria, producing secondary malignant **retrogression**; the process

II. Parasitic Theory.

being exactly similar to the actions of staphylococci or streptococci or treponema pallida, or tubercle bacillus, which produce suppuration or gumma or caseation or other similar changes, in a granulomata, acted upon by a secondary contamination.

The manner in which carcinoma or sarcoma resembles some retrogressive lesions such as granulomata, in their mode of onset, history, cachexia, their mode of producing secondary growths and metastases, and their clinical phases, are so characteristic, that their very nature suggests that these varieties of lesions are caused by **micro-parasites** of some kind.

All researches devoted to the elucidation of such ætiology have been made for cancer, and there is yet no suggestion or evidence of doubt as to the non-infective nature of non-malignant neoplasm.

(i) Bacteria.

(i) BACTERIA were sought for, and Doyen described a micrococcus in carcinoma called micrococcus Neoformans. *Vide* Vol. 1. It is not proved, nor accepted to be the causative organism, although it produces some irritative phases resembling a neoplasm.

(ii) Fungoid Bodies or Cancer Bodies.

(ii) FUNGOID bodies called Russel's bodies, and Ruffer or Plimmer's bodies were described and demonstrated in cancer cells. They were suggested to be parasitic protozoa, probably sporozoa. These bodies are found in abundance in the growing portion of a rapidly progressing carcinoma. These are not proved or accepted to be the actual causative factor. They are often described as **cancer bodies**.

(iii) Plasmodial structure.

(iii) PLASMODIAL structures related to organisms belonging to the Myxomycetes have been described, which met with a similar fate.

(iv) Protozoa.

(iv) PROTOZOE such as the coccidium of rabbit coccidiosis, produce epithelial proliferation similar to granuloma, or tubercle, but they do not produce carcinoma. Their power of exciting the epithelial proliferation, gives a probable clue, that perhaps, the causative organism of cancer may be of some kind of protozoa.

All attempts to stamp a protozoon as a causal agent of cancer have failed.

(v) YEAST-like forms have been isolated and by inoculation apparent malignant neoplasm has been successfully produced. But the newly-formed growth is found to resemble granulomata and they are demonstrated not to be carcinomata, on close critical examination.

(v) Yeast-like forms.

(vi) STREPTOTHRIX-like forms have been isolated from malignant growths, but they are demonstrated by other observers as merely secondary saprophytic invaders.

(vi) Streptothrix-like forms.

(vii) SPIROCHÆTES have been detected in mouse carcinoma. There is no evidence to suggest that they are the causative factor. This type of spirochætes cannot be stained but can be demonstrated by silver impregnation method of Levaditi, like those of syphilis. Similarly Demodex in cancer of the skin, Nematodes in cancer of the breast of the mouse or stomach of the rats, parasites in cockroaches have all pretended their claims with equal results, before the jury of observers.

(vii) Spirochætes.

None of the above theories based on the above types of parasitic invasion, has yet been accepted. But some very recent observers are demonstrating that Neoplasms are of **infective** origin. It is too early to state anything in this connection. So far as parasitical infection is concerned, some observers state, that it is possible, that the virus is intra-cellular; or an endo-toxin, induced by the irritation caused by the yet undetected parasites themselves, or some virus ultra-microscopic in nature, of living or dead parasites or both, excites the cells to start neoplastic multiplication, the actual genesis of which is not yet known.

Recent experiments by Gye proves that all malignant neoplasms contain a virus which is ultra-microscopic and capable of cultivation. This virus probably remains within the cancer cells. This school suggests that cancer

Gye's Experiments. Ultra-microscopic Theory.

Two distinct factors:

*Viz.*,

(1) An Irritation, or an Intrinsic and Specific.

(2) A virus, or an Extrinsic non-specific.

is produced by two distinct factors, *viz.*, (1) an intrinsic specific factor, which is the product of chronic irritation entirely produced by the cells; (2) a virus which is common to all tumours but introduced from without. The first one is a definitely specific factor and it is a chemical substance, which varies from tumour to tumour, and from tissue to tissue, whilst the virus is non-specific.

The parasitic theory has been adopted by many observers, but the weight of universal acceptance is still definitely wanting.

The other theories such as the Embryonic Hypothesis, the Post-Embryonic Hypothesis, theories based on Cytological observations, effects of inoculation, effects of transplantation, etc., are interesting from research and classical points of view. They are enumerated to help practical clinical observations if such occasion would demand.

### III. Embryonic Hypothesis.

#### 1. Conheim's Embryonic Rest.

### III. EMBRYONIC HYPOTHESIS.

1. CONHEIM'S THEORY OF EMBRYONIC REST.—This was originated by Virchow in some particular instances only, but was broadened and generalized by Conheim. By this, Conheim ascribed the genesis of neoplasms to latent embryonic rudiments. In normal development, cells allotted for the formation of certain organs may remain latent and undeveloped for a considerable time. When in the economy of the metabolism or under the stress of normal physiologic developmental demands, active part and function of that organ are required, their cells again start to proliferate, multiply, and functionate. The same phenomenon may occur even in some isolated cells of any of the three embryonic layers, exciting them to develop into neoplasms. During the life time of the organism, groups of such isolated cells remaining *at rest* may lie dormant and inactive, in their own tissue of origin or transplanted or transferred elsewhere, not being required to be developed into proper

histological tissues. Some kind of irritation or injury or as a matter of fact any stimulus or alteration of nourishment may excite or stimulate them to proliferate and form new growths at any later stages or phases of life.

Instances of such Embryonic Rests giving rise to the development of neoplasms are:—

Instances of Embryonic Rests giving rise to Neoplastic growths.

(a) The malignant tumours at the edges of the orifices of the body; *e.g.*, the muco-cutaneous junction of the lips and anus.

(b) Multiple Chondroma of the hand, met with in rickety patients. These are perhaps due to accidental inclusion of cartilage cells in the growing bone.

(c) Chondroma found in the lungs developing from bronchial cartilage, may be produced by the same cause.

(d) In adenomata of the testis and parotid glands, cartilage is frequently found. This is perhaps due to inclusion of embryonic tissues and cells from which cartilage is developed. In such parotid tumours they may come from the Meckel's cartilage. In the case of testis they may come from the spinal cord, or they are Teratomata.

(e) Chondroma may occur in the spinal cord arising from remnants of notochord.

(f) Growths in the kidney from adrenalin rests.

(g) Tumours of striped muscle observed in the kidney is probably due to inclusion of such cells in the developing kidney.

(h) Tumours and dermoid cysts, at or near the dividing line of two different types of tissues, *e.g.*, cancer of the lip at the junction of the cutis and mucosa, or at the anus as stated above, cervix uteri, branchial cleft, cysts in the neck, etc., that is to say, where the squamous epithelium unites with the spheroidal or cuboidal or glandular epithelium.

(i) Dermoids in the ovary and testis. These are attributed to the Germ-cell theory described below.

(j) 'Tumours arising from congenital soft warts or moles or maternal nævi, where embryonic tissues are demonstrated, in malignant transformation.

Whether parasitical, or irritative, whatever may be accepted to be the possible cause of neoplasms, no other theory but Conheim's, explains the above curious growths so satisfactorily. It is not explained what is that actual factor, which suddenly excites or stimulates the activity of a long dormant group of cells.

2. Beard's theory of Embryonic germ cells.

2. BEARD'S GERM-CELL THEORY.—This theory ascribes, that a body cell may fertilize another body cell in the same individual, and the relation of such a pathological growth with the host is exactly similar to the relation of physiological foetus to the mother. Both these conditions may be understood to arise from similar origin, with the only difference that one is a sexual and the other is an asexual development; that is to say, the cell from which a neoplasm develops may be a true sexual cell. A sexual cell may get displaced which may start growing at any place wherever lodged. Beard says, "In the history of the race, there has been a reduction in the number of actual normal embryos arising, but a persistence of such 'embryonic germ' cells. A malignant tumour, either cancer or sarcoma, is nothing more than an irresponsible trophoblast or chorion, the asexual generation, which in every normal development, is the forerunner of an embryo." (Hewlett).

This theory fits best to explain the genesis of many types of Teratomas. We shall discuss it in greater detail in connection with Teratomata.

3. Thiersch's Theory of Lost Balance, or Alteration of Tissue-Tension.

3. THIERSCH'S LOST BALANCE THEORY OR ALTERATION OF TISSUE TENSION.—This theory propounds, that in normal condition a tug of war exists between various tissues, *e.g.*, connective tissue and the epithelium, in order to keep each other in check by which they control and balance the growth of each other and they exist in a state of equilibrium. Their balance may be thrown out of

gear by early senescence, especially the connective tissue allowing the epithelial tissue to proliferate, unchecked. Owing to these degenerative changes in the connective tissue cells or the retrogression of the sub-epithelial tissues, which the theory presupposes, the epithelial tissue acquires or maintains a special vigour of unlimited multiplication and thus invades the connective tissues, and produce carcinoma. But this theory does not mention what happens in connective tissue tumours, and on whom they win in the tug of war. It is also inadequate to explain the infiltrative powers of malignant growths or their powers of secondary metastatic colonies. It does not explain why in such senescence, every body does not acquire a growth because senescence is a common factor.

#### IV. POST-EMBRYONIC HYPOTHESIS.

IV. Post Embryonic Hypothesis.

The above are all theories which are concerned in the anomaly of embryonic tissues. The following theories attribute the cause not so much to the embryonic character of the cells but their detachment or separation of the individual cell or groups of cells from the normal positions and controlling influence. These are:—

##### 1. RIBBERT'S THEORY OF DISTURBED EQUILIBRIUM OR THEORY OF GROWTH LIBERATION.

—Ribbert asserts, that all neoplasms are caused by two factors, namely, (a) separation of individual cells or groups of cells from their normal position, and (b) the loss of control or cessation of influences of the neighbours allowing the displaced cells to multiply autonomously, at the expense of the host. In such circumstances, they simulate the actions of parasites and behave as such.

1. Ribbert's theory of Disturbed Equilibrium or Theory of Growth Liberation.

When an organ is fully developed an equilibrium is established between the reciprocal influences, exerted by the cell elements constituting the organ, exercising on one another. Therefore, the more is the departure from the normal, the more the disturbance of equilibrium results and consequently the more proliferation follows.

There is, of course, **something** or some energy which controls cell-proliferation, be it either for growth as in intra-uterine life or for repair and maintenance as happens in extra-uterine life. But, it is not yet found out, what is that something. Internal secretion, nutritive limitation, mutual pressure, chemical action, physical influence, have all been suggested. The main point in Ribbert's theory in connection with epithelioma is, that he believes, that the fundamental fact is the detached condition of the epithelial cell-groups by the growth of connective tissue as a direct effect of chronic inflammation and subsequent indeterminate proliferation of these isolated epithelial cell-group.

2. Adami's  
Theory of  
Habit of  
Growth.

2. ADAMI'S THEORY OF HABIT OF GROWTH.

—We have pointed out under the chapters of Regeneration and Repair that the cellular life and activity depend on three factors; *viz.*, Nutrition, Reproduction and Function. Growth of a part or organ depends on the amount of Reproduction by multiplication, discharging the energy by the manifestation of Function.

Such reproduction takes place by active cell-division and cell-proliferation, or in other words, are the result of metabolic processes of life, which are controlled by the nucleus which plays the predominant part in both proliferation, as well as performance of function.

Such active cell-division would occur only under two conditions, *viz.*:—(1) by utilizing the **assimilated materials**, and (2) by the energy stored up by such assimilation. That is to say, during the performance of the animal's **specific function**, by the utilization of its food or nutriment and energy, active cell-division takes place. This may also happen where some stimulation from without results in increased assimilation.

Intrinsic  
cause of  
Cell-  
division.

It is then clear that increased assimilation and storage of increased energy will result in increased cell-division. This of course constitutes to be an **intrinsic cause** of increased cell-division.

Normally, this increase cannot be prolonged indefinitely inspite of increased *intrinsic* cause, as there are so many *extrinsic* inhibiting or repressive factors from outside, viz. ; (1) surrounding physical relationship, (2) the tension exerted upon the cells, or in other words the factor of Equilibrium of Tension. This latter factor is an **extrinsic cause**, which according to circumstances such as chronic inflammation, etc., or of foreign relationship, would exert and excite cell-division or inhibit such cell-proliferation, that is to say, act as the external circumstances would indicate, or warrant. Now then the distribution of the product of assimilation is pivoting on *work* or function on one side, and *growth* or proliferation on the other. In a fully differentiated cell under normal conditions, practically all the available energy is utilized for the purpose of *work*, and little is left for proliferation or *growth* ; the two items on demand being absolutely antagonistic to one another. It is only the **anaplastic** cells or where retrogression in the cells has occurred where **reverse** is the case that is *growth takes precedence of function*.

Extrinsic  
cause of  
Cell-  
division.

If, for *any cause*, the cells are diverted from the influence of inhibition and their proper extrinsic factors, and stimulated to proliferative activity, and put in circumstances under the influence of increased **intrinsic** function, inducing or stimulating such proliferative activity, they go on growing or a "habit of growth" is set up.

The greater the amount of cell-proliferation set up, the greater is the tendency to get away and divert from **extrinsic** control. The **growth at once adopts a purposeless militarism**, and the cells continue to multiply, resulting in a purposeless, functionless, neoplastic army of growth. This is Adami's theory of "Habit of Growth", and he bases the whole subject on defining the cause of **anaplasia**, which is such a striking feature in all malignant neoplasms.

V. Cyto-  
logical  
observa-  
tion and  
Hypothesis.

V. CYTOLOGICAL OBSERVATIONS AND  
THEORIES BASED ON THEM.

1. Theory  
of Mitotic  
Division.

1. THEORY OF MITOTIC DIVISION.

This was propounded by Farmer, Moore and Walker. These observers pointed out, that in ordinary physiological cell-division, the following stages are observed; viz. :—

Physiologi-  
cal cell-  
division by  
Mitosis.

1. Resting condition of mother nucleus.
2. Close skein of fine filaments.
3. Open skein of thicker filaments. Spindle appears.
4. Movement of V-shaped chromosomes to middle of nucleus. Each splits into two sister threads, in its own longitudinal axis.

This stage should be noted to be the most important part of the observation. The number of such chromosomes is always **constant for the same species** of animal, but it always **differs in different species**. In human cells, there are twenty-four chromosomes in the dividing somatic cells. They split in longitudinal axis.

5. Stellate arrangement of V-shaped filaments at equator of spindle.

6. Separation of cleft filaments, and movement along fibres of spindle.

7. Conveyance of V-filaments to opposite poles.

8. Open skein in daughter nucleus.

9. Close skein in daughter nucleus.

10. Resting condition of daughter nucleus.

The above is the form of somatic mitosis. As division proceeds on, a stage is reached at which a differentiation is observed. This differentiation is called **heterotype mitosis**. It is characterized by the following deviations; viz. :—

(a) The chromosomes are not well marked.

(b) The shape is not so distinctly V-shaped, but exhibit a bunched appearance.

(c) The number is also decreased to half the original standard.

(d) They divide transversely and not longitudinally.

(e) They assume the forms of loops or rings, and not like the shape of V.

Subsequently, they retain the reduced number, but regain the normal process. This post-heterotype mitosis is called Homotype Mitosis, or reduction division. The cells which exhibit reduction division, constitute the sexual or reproductive cells. Tissues exhibiting such cells are called **gametogenic**.

Farmer, Moore, and Walker pointed out, that, at the growing margin of carcinoma, many cells exhibit the phase of homotype mitosis. Thus, in this respect, the transformation resembles the sexual or reproductive cells and they suggest the malignant process as being due to a conversion of somatic cells into the reproductive type. The tissues formed of such cells in the pathological process may be termed **gametoid**. Such gametoid tissue and reduction division could be demonstrated in **malignant neoplasms** only.

These observers also demonstrated that structures similar to the **cancer bodies** of Ruffer and Plimner which were described as parasites, *vide* above, are evident in normal reproductive tissues. It is a curious irony of fate that such **structures** and **bodies** are present in normal reproductive cells and cancerous growths only, but they are never observed in non-malignant growths.

Moor-Walker's demonstration disproves the parasitic theory of Ruffer-Plimner. At the same time, it lends support to the Germ-cell theory of Beard.

This does not explain what factor produces the gametoid cells. As in plants, irritation excites such heterotype mitosis, it seems to be possible that it is **irritation** which brings about this heterotype mitosis also.

Gameto-  
genic  
Tissues

Presence of  
Ruffer's  
Cancer  
bodies like  
structure  
in normal  
sexual  
cells.

Ruffer's  
'Cancer-  
Bodies'  
are not  
parasites.  
Irritation  
is the  
factor to  
produce  
Heterotype  
mitosis.

2. Other alterations in the cells of Malignant growths.

## 2. OTHER ALTERATIONS IN THE CELLS.

Both in sarcoma as well as in carcinoma the Altmann's granules are found to disappear from the cells. These granules are normally present in the cytoplasm, excepting in the squamous epithelium and part of the kidney tubules.

Cells of malignant tissues fixed in formol and then stained, do not exhibit these granules, although they manifest themselves in other situations being treated similarly.

## EFFECTS OF TRANSPLANTATION AND INOCULATION.

Effects of Transplantation and Inoculation.

The following facts are deduced by transplanting tumour in animals especially mice. In practical surgery, the datas help us in studying the clinical features of our cases and guide us in taking particular care during operation. No undue susceptibility or liability has ever been demonstrated to be present amongst surgeons, pathologists, nurses etc. The datas are:—

(i) In the mouse, all kinds of growths can be transplanted more or less.

(ii) The percentage of successful transplantation varies with the different kinds of neoplasms.

(iii) Spontaneous neoplasm sometimes undergo spontaneous absorption.

(iv) If such a mouse which gets a spontaneous tumour, be inoculated with a portion of its own tumour it is practically always successful. But tumours implanted into **other** spontaneously affected mice fail in most of these cases.

(v) This proves that "an animal is very susceptible to its **own** tumour, but is no more susceptible to the tumours of other animals than are normal animals; each tumour is peculiarly and genetically related to the individual in which it arises." (Hewlett).

(vi) If it is an inoculated mouse, the transplanted graft of a particular kind of tumour undergoes absorption, such a mouse becomes to a certain extent **insusceptible to inoculation** with such tumours, but is still **susceptible to the spontaneous development** of another kind of malignant growth.

(vii) So specific are the tumours, that if one kind of animal be inoculated with tumours developed from another kind of animal, the graft of the latter tumour does not grow properly in the former animal, but very soon takes to involution. This is so specific that the tumours of one race grow with considerable difficulty in another race.

If this fact be true amongst animals, it may be possible in mankind also. This gives a clue why amongst different races of man, the same kind of tumours are found to develop with different features.

"This specificity of tumour growth is correlated with specificity of biological properties." (Hewlett).

(viii) Injection of normal tissues induces a considerable degree of insusceptibility, for instance injection of skin would protect against carcinoma, and the resisting immunity persists several weeks, even up to three months.

(ix) It is only the inoculation of the **living cells** which resists the development of transplanted tumours. Those living cells may be tumour cells or normal cells. Disintegrated and **dead cells** do **not** exert **any** influence.

(x) So far as inoculation is concerned, if the graft is successful, malignant neoplasms grow unceasingly; but non-malignant growths, when injected young, may grow for a limited time, which in all cases, if non-malignant, would eventually cease to grow.

(xi) In a large number of cases, the first inoculation is successful, but in the majority of instances the second inoculation fails. The reason as explained by Ehrlich, is, that every animal possesses a certain yet unknown substance called by him as X-stuff. This X-

stuff is required to enable the neoplasm to secure the necessary nutrition for its growth. If all this X-stuff available in the subject is used up by the growth in the first inoculation, the second one gets no more of it to grow, and an immunity is thus produced for the time being. Ehrlich termed this kind of immunity as **atreptic immunity**. This is caused, as he says, by the deficiency of the X-stuff. A distinct ebb and tide in all growths is observed, which is exhibited by alternate activity of proliferation or dormant condition and regression in all tumours.

(xii) "Cancer and non-cancer tendencies are unit characters which can be implanted in any species or eliminated permanently and completely. There is, therefore, a ready and certain genetic road of escape from cancer for the individual and the race."

### INFLUENCE OF NERVES IN THE DEVELOPMENT OF CANCER.

Influence  
of Nerves.

Rodent ulcer and some forms of cutaneous carcinoma, seem to show a definite line of development in the areas of distribution of certain nerves.

Factors in  
Blood in  
relation to  
malignant  
growth.  
Glucose is  
increased.  
Lipolytic  
power.

### BLOOD IN RELATION TO MALIGNANT GROWTHS.

Patients suffering from Carcinoma show a distinct increase of glucose in the blood.

Anti-  
tryptic  
action.

Normal blood possesses a marked degree of anti-tryptic action. This power of inhibiting the production of trypsin is observed to be increased to double, or sometimes more, in the cases suffering from carcinoma and sarcoma.

Increase of  
Alkalinity.

Increased alkalinity of the lymph stimulates cell growth, and if this condition remains long continued,

may lead to excessive cell-division, and may induce the cell-growth to malignancy.

It is noteworthy that irritation, inflammation, old age, early senescence etc., are all accompanied by increased alkalinity of the lymph.

Change in the distribution of salts in the plasma, whereby the alkalinity of the latter is increased, may cause diminution in the secretion of the gastric hydrochloric acid. This condition has been associated with the development of carcinoma, especially of the colon.

Causing  
Hypo-  
chlorhydria.

POTASSIUM salts increase enormously in the blood and tissues of cancer cases. All rapid growths imply an increased demand for **calcium** salt from the blood of the host. This rule holds good equally in physiological as well as in pathological conditions, *e.g.*, there is an enormous demand for calcium salts from the blood in pregnancy, owing to rapid cellular multiplication. The important point is that there is a marked decrease in calcium salts of the blood in cancerous cases. Such decrease of calcium salts is also observed in the blood of pregnant women. Thus, potassium salts are antagonistic to calcium.

Increase of  
Potassium  
Salts.

Decrease of  
Calcium  
Salts.

As age advances from adult to middle period of life, there follows a progressive decrease in calcium content of the blood. The calcium gets bound in the tissues and free calcium becomes less and less available. This deficiency in calcium salts becomes pronounced in the cases of cancer. We shall return to the subject in a subsequent chapter in connection with clinical observations.

Potassium  
salts are  
antagonis-  
tic to cal-  
cium salts.

## CANCER HOUSES AND CANCER COUNTRIES.

Cancer  
Houses  
and Cancer  
countries.

Existence of particular houses where members of the family and occupiers had become the victims of

cancer, has been reported. Such local incidence of cancer points to its infective nature.

Incidence of cancer with reference to :—

1. AGE.
2. SEX.
3. HABIT AND MODE OF LIFE.
4. FOOD.
5. CONSTIPATION.
6. HABITAT, locality, geographical distribution, etc.
7. CIVILIZATION.
8. HEREDITY,

Will be described in greater detail in connection with malignant neoplasms.

#### SUMMARY.

The consistency of growth and the utility to serve some purpose in the working of the system are the features in Hypertrophy. Neoplasm means 'New elementary life.' Their ways of growth is peculiar, *e.g.* :—

- (i) In size.
- (ii) In shape and form.
- (iii) The surface.
- (iv) Consistence.
- (v) Colour.
- (vi) Structure.
- (vii) The progress of growth. Malignant and Benign.
- (viii) Their mode of invasion.
- (ix) Their history.

History of a Benign Neoplasm and characters. They grow by Expansion. History of a Malignant Growth. They grow by Permeation and Infiltration. Two primary parts.

(a) The Parenchyma or the proper Neoplastic cells.

(b) The connective tissue stroma or Matrix.

The changes in the Blood vessels, Nerves, Lymphatics. Function of the affected tissue. Terminology. The present nomenclature does not help the classification. Pathological Classifications. Adami's Scheme Modified. Powell-White's classification. Blastoma is a better term. Embryonic Development. The three Embryonic Layers. Table T. T. and A. C. T. Powell-White's method of Classification.

**A. Histiomata or TISSUE Tumours.****I. MESOBLASTIC.**

- (1) Endothelial Tissue.
- (2) Connective Tissue.
- (3) Muscular Tissue.

**II. EPIBLASTIC.**

- (1) Nerve Tissue.
- (2) Epithelial Tissue.

**B. Cytomata or CELL Tumours.****I. EPIBLASTIC CELLS.****II. MESOBLASTIC CELLS.**

- (1) Endothelial Cells.
- (2) Connective Tissue Cells.
- (3) Lymphoid Cells.
- (4) Muscle Cells.
- (5) Mixture of Indifferent cells.

**III. PIGMENTED TUMOURS.****IV. HYPERNEPHROMATA.****V. ORGANOMATA. Teratoma.**

Clinical Classification and Pathological Classification. Surgeons use the terms Benign and Malignant on clinical observations. Non-Malignant and Malignant. The Broad Features of both. Table. Etiology.

**I. VIRCHOW'S THEORY OF IRRITATION.**

- (i) Mechanical Irritation.
- (ii) Thermal Irritation.
- (iii) Chemical Irritation.
- (iv) X-rays Irritation.

**II. PARASITIC THEORY.**

- (i) Bacteria.
- (ii) Fungoid Bodies or Cancer Bodies.
- (iii) Plasmodial structure.
- (iv) Protozoa.
- (v) Yeast-like forms.
- (vi) Streptothrix-like forms.
- (vii) Spirochaetes.

Gye's Experiments. Ultra microscopic theory. Two distinct factors. *Viz.* :—

- (1) An Irritation, or an Intrinsic and Specific.
- (2) A Virus, or an Extrinsic non-specific.

## III. EMBRYONIC HYPOTHESIS.

- (1) Conheim's Embryonic Rest. Instances of Embryonic Rests giving rise to Neoplastic growths.
- (2) Beard's theory of Embryonic germ cells.
- (3) Thiersch's Theory of Lost Balance, or Alteration of Tissue-Tension.

## IV. POST EMBRYONIC HYPOTHESIS.

- (1) Ribbert's theory of Disturbed Equilibrium, or Theory of Growth Liberation
- (2) Adami's Theory of Habit of Growth.

Intrinsic cause of Cell-division. Extrinsic cause of cell-division.

## V. CYTOLOGICAL OBSERVATION HYPOTHESIS.

## 1. Theory of Mitotic Division.

Physiological cell-division by Mitosis. Gametogenic Tissues. Presence of Ruffer's Cancer body-like structure in normal sexual cells. Ruffer's 'Cancer-Bodies' are not parasites. Irritation is the factor to produce Heterotype mitosis.

## 2. Other alterations in the cells of Malignant growths.

Effects of Transplantation and Inoculation. Influence of Nerves. Factors in blood in relation to malignant growths. Glucose is increased. Lipolytic power. Anti-tryptic action. Increase of alkalinity. Causing Hypo-chlorhydria. Increase of potassium salts. Decrease of calcium salts. Potassium salts are antagonistic to calcium salts. Cancer houses, and Cancer countries.

## CHAPTER III.

### NON-MALIGNANT NEOPLASMS.

We have already stated that from clinical point of view it is convenient to classify Neoplasms into two broad groups, *viz.*, non-malignant, and malignant. This is convenient from the view point of our clinical studies, and it makes the subject easy to understand. We shall therefore describe the morbid anatomy of tumours under those two groups. Their broad differentiating features have already been described.

In describing the Non-malignant Blastomata serially, we shall for all practical purposes try to follow the following two great divisions, *viz.* :—

A. Hylomata, or Connective Tissue Tumours.

A. Hylo-  
mata.

B. Lepidomata, or Epithelial Tissue Tumours.

B. Lepido-  
mata.

The broad histological features which help us to differentiate the two kinds of tumours are the following :—

(1) In all lepidic or epithelial tissues whatever the arrangement may be whether the epithelial tissue spreads over a surface and works as a protecting tissue, or a covering membrane, or lining an organs; or collected together to form a solid organ, the epithelial cells constituting the tissue remain always in **absolute contact** with one another; there being **no intercellular** substance present such as are seen universally in all connective or hylc tissues.

Their  
differenti-  
ating  
features.

(2) The collection or **groups** of cells in epithelial tissues are supported by **stroma** or scaffolding of connective tissue. This connective tissue scaffold works as a support for that particular group, but never interferes with any particular distinctive arrangement the cells in the group assume to adopt. The connective tissue simply works for services; or in other words they supply *labour* to the more organized group.

(3) The services which the connective tissue supplies to the epithelial tissue consist of:—

(i) Physical support in the shape of preparing a ready scaffolding or skeleton.

(ii) Physiological support in the shape of supplying nutrition and maintenance, which is of vital importance to the epithelial tissue; all the blood and lymph transported to it being contained in the vessels constituted of the stroma supplied by the connective tissue; any interference of which would produce starvation and degeneration of the centrally situated cells in the group of the epithelial cells.

(iii) Carriers of metabolic waste.

(4) It is thus evident that epithelial tissue *cannot exist* without the presence of the connective tissue; and it is obvious then that it can not develop or proliferate without the development *pari passu* of the connective tissue, which necessarily means that the latter must keep pace with that of the epithelium.

(5) Thus the growth of the connective tissue is purely **subservient** to that of the proliferation of the epithelium. And in a particular Histioma, either composed of hylic or connective tissue or composed of lepidic, that is, epithelial tissue, where both elements progress we are to distinguish one or the other element as the *predominant* partner, to determine its integrity.

(6) In view of this intimate relationship between the hylic and lepidic tissues in non-malignant Lepidomata, the latter are sometimes described as **fibro-epithelial** tumours.

(7) On the other hand we must remember that in pure **hylomata** no such trouble as the above arises as they do not depend upon the higher organized class, as they prepare their own food and make their own house or scaffold.

The non-malignant tumours we are going to describe in this chapter are:—

**A. HYLOMATA.**

The list  
of Non-  
malignant  
Tumours.

**1. MESOBLASTIC ORIGIN.**

- I. Lipoma.
- II. Fibroma.
- III. Chondroma.
- IV. Osteoma.
- V. Myxoma.
- VI. Myeloma.
- VII. Lymphoma.
- VIII. Myoma.
- IX. Angioma.

**2. EPIBLASTIC ORIGIN.**

- X. Glioma.
- XI. Neuroma.

**B. LEPIDOMATA.**

- I. Endothelioma.
- II. Epithelioma (Non-malignant) Ziegler.

*Or*

Epithelial-tissue tumours.

- 1. Papilloma.
- 2. Adenoma.

**A. HYLOMATA.**

A. Hylo-  
mata.

**I. LIPOMA.**

1. Lipoma

The commonest benign tumour met with and well known even to lay people is Lipoma. Lipoma is a non-malignant slowly progressing growth composed of adipose tissue. It is formed of fibro-cellular tissue infiltrated with fat. On naked eye or macroscopical examination it differs in no way from normal adipose tissue. On microscopical examination, the fat cells appear to be comparatively of bigger size than normal fat cells of adult type.

Shape,  
Single or  
Multiple.

In **shape**, a Lipoma generally appears rounded and flat, usually circumscribed and often lobulated. It is sometimes multiple.

Surface.  
Consistence.  
Colour.  
Capsule.

The **surface**, of a lipomatous tumour is smooth, its *consistence* is doughy and on firm pressure it feels elastic. It is somewhat of paler *colour* than the surrounding fat.

Lipomatosis.

In some instances of **lipomatosis** which are blastomatoid in nature it is found to be uncircumscribed and diffuse. This condition being an exception from benign tumours, which are generally provided with a definite capsule, helps us in diagnosing it to be of the nature of diffuse lipomatous blastomatoid infiltration. It is met with growing under the chin or at the back of the neck. A typical Lipoma is always surrounded by a delicate fibrous capsule, and is thus sharply encapsuled from which coarser septa pass inwards between the lobules.

Size.

In **size**, it may be large or small; often growing very large, as big as a melon; or sometimes remaining as a small nodule only of the size of an almond or walnut.

Situations.

The **situations**.—Lipomata are particularly and frequently seen at places which usually undergo much pressure or friction in our ordinary life; such as the subcutaneous tissues of the shoulder, neck, forehead, back, axilla, groin, &c. They also grow in the adipose layers of the viscera; *e.g.*, appendices epiploicæ, or larynx, periosteum of long bones, synovial fringes of joints, and sometimes at other situations, *e.g.*, mammary glands, uterus, liver, meninges of the brain and spinal cord.

Structure.

**STRUCTURE**.—On section, the tumour is found to consist of small lobules interspersed with fibrous septa which are filled with fat and in the walls of which the blood vessels run. Microscopically, the growth exhibits the ordinary structure of adipose tissue the cells of which are comparatively of bigger size. Between the cells are sometimes detected some myxomatous tissue. It is not as freely supplied by blood vessels as the normal adipose tissues, and it is for this deficiency of blood supply a

Lipoma looks paler than the normal surroundings as described above. The fat cells and fat globules in a neoplastic growth are generally larger than those in the ordinary normal fatty tissue in comparison.

A microscopical section exhibits only some vacant spaces bounded by strands of fibrous tissue as the fat globules from the groups of cells separated by these delicate strands are dissolved away by xylol in the staining process. By examining such a section only, it is not possible to say if it were from a tumour. The newly formed blood vessels are well-formed, and are surrounded by abundant fibrous sheath.

It must be remembered that Lipoma is not Adiposis, which arises from *metabolic defect* usually found in the obese and is a **general** condition; whereas Lipoma is a **localized growth**, always encapsuled. The fat cells in a Lipoma seen under the microscope are of the adult type, and as already mentioned they are slightly larger than the cells of the normal adipose tissue. Occasionally the cells may approximate closely to the foetal type of fat cells, exhibiting the fat in the form of many globules in the cytoplasm instead of a single large one occupying the whole area of the cell protoplasm. It must be recollected that Blastomatoid Lipomatosis is not adiposis.

PROGRESS.—They may grow, remain stationary, or degenerate. The degenerations liable to occur are, (1) Myxomatous change, (2) Ulceration, (3) Calcification, (4) Liquefaction with the production of Cyst; and (5) Sarcomatous change or other malignant transformations.

The following **clinical varieties** of Lipoma are met with :—

Clinical  
Varieties.

#### A. BLASTOMATA :—

A. Blastomata.

(i) Localized Subcutaneous Lipoma.

The commonest form.

(ii) Deep Inter-muscular Lipomata or Subfascial Lipomata.

(iii) Parosteal Lipomata.

- (iv) Pericranial Lipomata.
- (v) Subserous Lipomata.
- (vi) Fatty Hernia of the Linea Alba.
- (vii) Sub-synovial Lipomata.
- (viii) Submucous Lipomata.
- (ix) Painful Lipoma of the foot, or 'Tubby's disease.
- (x) Nævo-Lipoma.

#### B. Blastomatoïds.

#### B. BLASTOMATOIDS :—

- (i) Diffuse Subcutaneous Lipomatosis.
- (ii) Adiposis Dolorosa.

#### C. Mixtures and Transformations.

#### C. MIXED TUMOURS and MALIGNANT TRANSFORMATIONS :—

- (i) Fibro-Lipomata.
- (ii) Myxo-Lipomata.
- (iii) Angeio-Lipomata.
- (iv) Growth of Sarcomatous Tissue.
- (v) As a common constituent of Teratoma.

All the above are described in a fuller detail below.

#### (i) Localized Subcutaneous Lipoma.

#### (i) LOCALIZED SUBCUTANEOUS LIPOMA.

This variety of Lipoma is typically rounded in outline ; it is often lobulated, the growth is soft and doughy and feels semifluctuating, resembling a cold abscess. The mass can be held by the fingers and the thumb and moved from side to side. While gliding in this way, the overlying skin dimples which is drawn in within the mass owing to the fibrous trabeculæ traversing from the capsule into the skin. This localized variety is provided with a definite capsule, is slow growing, and is freely movable. In the cases where occupation subjects the tumour to constant friction or pressure like a bursa, this movable character is lost, and the tumour soon becomes flattened and adherent to the surrounding and subjacent tissues and structures.

Lipoma of this nature is generally single but may become multiple, sometimes occurring in considerable numbers such as dozens. The latter condition develops more commonly on the trunk or the upper extremity. In the upper part of the thigh sometimes huge single, or less commonly multiple type, of Lipoma develops ; which may become pedunculated and pendulous looking like jack fruits hanging on the trunk of a jack fruit tree.

Difficulty may sometimes arise in diagnosis of Lipoma which is not sufficiently defined on the surface, although in the majority of the cases it is easy. For diagnosis of Tumours in general, *vide* chapter vi, of this volume. It is often confused with various cystic (sebaceous) and fluctuating swellings. Even from cold abscess it is sometimes difficult to differentiate. Fluctuation and swelling is common to both. Both may be lobulated. Heat, redness and pain are absent in both. Both may be equally mobile. In a chronic abscess, the swelling is less defined in outline and a cold abscess especially has a shelving margin and usually fixed to the deeper fascia. The skin over a cold abscess is quite free, or when it is not so it may be adherent to a large area, and produces no dimple while the mass is moved. The edge of the Lipoma can almost be grasped between the fingers and thumb, and on such an attempt being made it slips off the tips. The resonance, dullness, signs of fluctuation, &c., are all absent in a Lipoma. For a fuller description, *vide* chapter vi, of this volume.

TREATMENT of this type of Lipoma is very simple. Treatment  
Excision is the only course, and the operation is easy. Operation.  
The tumour is held by the left hand and by gripping the base tightly the skin over the mass is made tense. An incision is then carefully made on the middle line. While little pressure is exerted by the left hand, the right index may be swept between the fibrous capsule underneath the skin and the growth which may result in a slow shelling out of the mass. In the cases where the skin is adherent with the mass a careful dissection and manipulation may

be required. In some cases the whole mass with the skin requires to be excised out, the raw surface being subsequently covered by skin grafting.

(ii) Deep  
Inter-  
muscular  
Lipoma or  
Subfascial  
Lipoma.

### (ii) DEEP INTERMUSCULAR LIPOMA, OR SUBFASCIAL LIPOMA.

Lipoma sometimes develops in the deeper tissues in the intermuscular spaces. They are often lobulated and encapsuled, although both these features may remain clinically absent, being masked by the superjacent tissues. They may also occur in the tendon sheaths of feet and hands resembling a ganglion closely.

Pathological characteristics of this type are just the same as those of the first type.

For clinical features and diagnosis *vide* chapter vi, of this volume. The type is sometimes very difficult to diagnose, and cases have been mistaken for sarcoma, cold abscess, periosteal growths, ganglion, etc., the real nature of which is confirmed on the operation table.

(iii) Paro-  
steal  
Lipoma.

### (iii) PAROSTEAL LIPOMA.

Lipoma sometimes grow from the outer surfaces of the periosteum on the vertex of the skull, described as Parosteal Lipoma.

The pathological characteristics do not differ in any way from the other types of lipomata. It is generally congenital, or it grows soon after birth.

Clinically, it is very difficult to diagnose them from sarcoma, gumma, or tuberculous abscess. For diagnosis, *vide* chapter vi.

Treatment.

TREATMENT.—Radical operation.

(iv) Peri-  
cranial  
Lipoma.

### (iv) PERICRANIAL LIPOMA.

This condition is exactly similar to the preceding type of lipoma. It is generally congenital. The peculiar feature

is that the cranium is often hollowed out beneath it. In some cases angioma develops in association with lipoma, giving it a characteristic feature of Nævo-lipoma or Angio-Lipoma. *Vide* Angioma.

TREATMENT is complete removal which is some- Treatment.  
times difficult owing to its close association with bone and nævoid vessels. Diathermy knives should always be preferred to ordinary ones to avoid excessive hæmorrhage.

### (v) SUBSEROUS LIPOMA.

(v) Sub-  
serous  
Lipomata.

Lipoma often grows in the subperitoneal fatty tissues such as the appendices epiploicæ of the large intestines or anywhere beneath the visceral and parietal peritoneum. Not infrequently, they develop in the lower part of the abdomen which may extend down to the crural or inguinal canals. By a continuous traction of the peritoneum these growths sometimes produce true femoral or inguinal herniæ which they precede sometimes. They also grow in the *submucous* tissue of stomach and intestines described below.

TREATMENT of extra-peritoneal cases is complete Treatment.  
excision. Operations are similar to those on hernia. *Vide* Regional Surgery.

### (vi) FATTY HERNIA OF THE LINEA ALBA.

(vi) Fatty  
Hernia of  
the Linea  
Alba.

Hernia of fatty masses or growths also occur through congenital or acquired openings in the linea alba or linea semilunaris. The growths are pedunculated in nature and are generally painful. This condition is described by various names, *viz.*, Epigastric Lipoma, Properitoneal Lipoma, Epigastric Hernia, Supra umbilical ventral Hernia, or Fatty Hernia of Linea Alba.

Its various  
other  
names.

TREATMENT consists of complete excision, and Treatment.  
closing of the apertures as done in an ordinary hernia.

### (vii) SUBSYNOVIAL LIPOMA.

(vii) Sub-  
synovial  
Lipoma.

Lipoma often develops in the fatty tissues of the synovial fringes of joints.

**Lipoma  
Arbores-  
cens.**

**LIPOMA ARBORESCENS** is a condition in which the villi overgrow in papillary synovitis springing from the reflection of synovial membrane which are loaded with fat. At times this development of fatty tissues may become considerable in the cases of Osteo-arthritis and Chronic Rheumatoid Arthritis, or sometimes mistaken for Baker's Cyst. For fuller description and treatment, *vide* volume II.

**(viii) Sub-  
mucous  
Lipoma.**

### **(viii) SUBMUCOUS LIPOMA.**

Submucous Lipoma may occur like subcutaneous lipoma under the mucous membrane of the mouth, pharynx, larynx, œsophagus and the intestinal tract. In these situations a lipoma not infrequently causes obstruction to the canal concerned, which may become fatal. In the small intestines when growing in the wall of the gut it may cause chronic intussusception. *Vide* vol. vi. It is a matter of extreme difficulty to diagnose a growth like this without operation. For the details of which, *Vide* vol. vi. Surgery of the regions concerned.

**(ix) Pain-  
ful Lipoma  
of the foot  
or Tubby's  
Disease.**

### **(ix) PAINFUL LIPOMA OF THE FOOT OR TUBBY'S DISEASE.**

This is a lipomatous growth which occurs on the inner side of the sole of the foot. The condition makes walking painful and the patients adopt a gesture of flat foot.

**TREATMENT.**—The only remedy is thorough operation. If the removal is not very thorough recurrence takes place.

**(x) Nævo-  
Lipoma.**

### **(x) NÆVOLIPOMA.**

Often Nævus grows in association with proliferation of adipose tissues when it is described as Lipoma. It will be described under Angeiomata. *Vide* below.

**B. BLASTOMATOIDS or LIPOMATOSIS.**

B. Blastomatooids or lipomatosis.

The Lipomatosis met with in connection with proliferation of fat cells working under the stimulus of reactive hyperplastic nature are the following :—

(i) Diffuse Subcutaneous Lipomatosis.

(ii) Adiposis Dolorosa in women or Dercum's Disease.

Both these above conditions not being Blastomata in the true sense of their pathological nature have already been described under Blastomatoids in the first chapter of this volume. *Vide* pages 19 and 20.

**C. MIXTURES AND TRANSFORMATIONS.**

C. Mixtures and Transformations. (xi) Fibromatous Myxomatous and Sarcomatous Lipoma.

**(xi) FIBROMATOUS, MYXOMATOUS AND SARCOMATOUS LIPOMA.**

Lipoma is a connective tissue tumour. In some instances other connective tissue basis may undergo modification. The fibrous stroma and septa constituted of connective tissue fibres may also increase out of proportion to the adipose tissue, and thus alter the tumour to become more fibrous than fatty. Such a condition is described as Fibro-Lipoma.

Similarly in some instances myxomatous tissue may dominate or mucoid degeneration may occur turning it into Myxomatous Lipoma, to be described later.

In some instances the type of the cells may turn to be embryonic and malignancy may set in, and turn a Lipoma into a Lipo-sarcoma, to be described later.

**XANTHOMA.**

Xanthoma.

The Xanthoma is a neoplasm occurring in over 90% of the cases on the eyelids. It is of yellowish, or sometimes golden yellow colour, soft in consistence, and generally grows only up to the size of a small almond, and half as thick, appearing like raised patches.

Xanthoma  
Diabeti-  
corum.

Microscopically, it is composed of embryonic adipose tissue of the nature of cholesterol or cholesterin fat.

Xanthoma affects several members of the same family or several generations of the same family. This affection in generalized form is often associated with jaundice, diabetes when called **Xanthoma-diabeticorum**, liver trouble, or chronic dyspepsia. It usually appears on the lower eyelids. Sometimes, Xanthoma develops as definite tumour in the serous membrane, tendon sheaths, and in other visceral parts of the body as in the pancreas of patients suffering from diabetes, or on the surface of the skin. In the latter instance, it involves the skin and the underlying soft parts as occurs in multiple neuro-fibromata, and even follows the distribution of the cutaneous nerves.

Presence of Xanthoma cells in myeloid tumour has been described in some rare cases, and mixed tumour composed of xanthomatous and myeloid tissue has been suggested, being described as Myeloxanthoma.

In rare instances tumour like form composed of Xanthoma may be found disseminated throughout the body after the manner of sarcoma. This condition of the tumour which appears to be a form of Fibroma consists of small spindle-celled cells or large polyhedral cells. These cells are filled with globules of yellow lipoid material.

Treatment consists in improving the general health and the condition of the liver. *Vide* cholesterin circulation in connection with cholelithiasis, Vol. II. It may be noted that the granules of lipoid material seen in Xanthoma consist of an ester of cholesterol and is similar to that which occurs in the "strawberry gall-bladder", corpus luteum of the ovary, and adrenal cortex.

II. Fib-  
roma.  
Consti-  
tution.

## II. FIBROMA.

Fibroma is a non-malignant neoplasm constituted of ordinary fibrous tissues. They are mainly connective tissue corpuscles flattened with branching processes and

arranged with white fibres in parallel wavy bundles. In some instances they arrange in layers like their arrangement in the cornea.

In naked eye **appearance** they seem to grow to various sizes and look like various structures. Some are known as simple warts ; some are polypoidal nodules ; some are more or less rounded or lobulated small tubercle-like masses ; some appear to be hard nodes or nodules ; on the other hand some may attain a considerable size weighing several pounds. They are generally pedunculated and pendulous. Appearance.

In **consistence** the tumour may become very hard and dense, or remain soft. The consistence varies according to the component tissues it is constituted, *viz.*, amount of œdema fluid that may be present or myxomatous degeneration that may occur or cellular or hard dense mass of fibres that it may contain. Consistence.

In **colour** these tumours are whitish grey, or pure white. Colour.

They are generally **multiple**, may be **single**, and are always encapsulated ; and are always localized. Multiple or single.

On **section**, if looked from a side, it presents an appearance of 'watered silk.' The tumour is encapsulated. The body of the mass contains very few blood vessels and owing to deficient supply of nutrition in the hardest part or the central part of the mass degenerative changes soon begin. Fluids of various nature may be found accumulated in the intercellular spaces, sometimes forming into cysts. In many of these areas myxomatous or mucoid degenerative changes are very markedly present. On section.

The **sites** where fibromata develop are most commonly the skin and next to the skin, the fasciæ, periosteum, tendons, tendon sheaths, sheaths of muscles, nerves, or solid organs, *e.g.*, uterus, breast, kidney, etc. That is to say, wherever fibrous tissue exists fibroma may develop, although a pure fibroma is of rare occurrence. According to situation in the body such growths may be loose and Sites.

succulent, *e.g.*, in the eye lids and scrotum; or dense and firm and even stony hard as in the tendons and ligaments.

Progress.

PROGRESS.—It grows slowly, but continually and degenerative and malignant changes, especially of myxomatous nature, often take place.

Clinical varieties.  
(i) Soft and  
(ii) Hard.

CLINICAL VARIETIES.—For the sake of convenience clinically fibromata are divided into two groups. *Viz.*, (i) Soft Fibromata and (ii) Hard Fibromata. There is no demarcation or essential difference between the two types as both the conditions may co-exist in the same tumour side by side, but clinically they are divided as such according to the resistance a tumour offers to pressure.

Their Microscopical and other peculiarities.

Microscopically the connective tissue *cells* are relatively more abundant in Soft Fibromata that is to say they are more **cellular**. The *fibres* actually dominate in the hard type or in other words they are more **fibrous**. The older the tissues the more fibrinous they are. In the soft parts the cells are distinctly elongated and spindle-shaped, being mostly **fibroblasts**, and bundles of **formed** fibrous tissue. The fibres in the harder portions are white fibrous tissue generally less wavy than normal, with a tendency to run in all directions or to form "whorls" or become interlacing. A small proportion of yellow elastic fibres may be present here and there as also some fat cells. The whorling and interlacing arrangements of **fibres** dominate in the hard type of Fibromata, and **cells** of spindle shape and elongated types constitute the main bulk of the soft variety. The younger the tissue the less fibrous they are. The formed blood vessels are of normal type; they are numerous in the early rapidly growing stage, and are situated in the **stroma**, which separates the tumour into lobules; but become more and more scanty and scarce later; especially in the hard type. The vacant spaces and areas, observed as filled with fine meshwork or with jelly like homogeneous matter, are areas of muroid or myxomatous degeneration.

Fibromata present such characteristic well formed condition of multiplication of the connective tissue fibres

that it is sometimes difficult to distinguish them from Hyperplasia. But the very fact of the bundles running in all directions exhibits the purposeless nature of the growth having no use of them.

Various other tissues may combine to proliferate and produce mixtures, *e.g.*, Neuro-fibroma, Fibromyoma, Fibro-lipoma, myxo-fibroma, etc.

(i) SOFT FIBROMATA develop as localized tumours of the subcutaneous fibro-cellular tissues. The more cellular these soft types are the more rapidly do they grow; as fibres of young tissues are always less fibrinous and the older the cells or the more time they take the more fibrous they become. Sometimes in their mode of such hurried development they resemble a sarcoma. When the growth is slow a soft fibroma often resembles a lipoma. The fibres of a soft fibroma tend to run in strands or bundles. Usually all cases of Soft Fibromata are not so rapidly growing. The blood supply is carried out in the **stroma** or cortex consisting of bands of connective tissue. Soft Fibroma.

The number of veins which develop amongst the lobules of the tumour are usually so numerous in a soft fibroma that it is their presence which is the primary cause of producing their softness. They bleed freely on very slight injury. This softness and tendency to bleeding may lead the beginner to a diagnosis of sarcoma in a rapidly developing case which may be experienced even under the microscope. The slowly growing ones may also be easily confused clinically with lipomata but fibromata are of much rarer occurrence. It must be remembered at the same time that old-standing cases of soft fibroma undoubtedly undergo malignant transformation especially to sarcomatous nature.

The soft fibromata commonly develop on the inner side of the upper arm close to the axilla, or at the thigh near the perineum. They are also met with, although rarely, in the labia, scrotum, on the scalp, and in the subcutaneous tissues of other parts of the body. Nasal polypi were formerly described as oedematous fibroma, but al- Other soft Fibromata.

though some of them may be of this nature, larger majority of nasal polypi are not of this kind. Various other factors may produce nasal polypi. *Vide* Regional Surgery. When Fibromas grow in the wall of a hollow viscus such as the rectum or alimentary canal they tend to project into the cavity as pedunculated polypi. Submucous Fibromata of the uterus described as uterine polpi are also of this nature. Subcutaneous and submucous varieties particularly take to this pedunculated polypus type.

(ii) **HARD FIBROMATA** develop as localized tumours in the sites where the fibrous tissue is hard and dense; *e.g.*, in the periosteum of the jaw bones in the form of Epulis, or better called Epulides; on the bones of the naso-pharynx as fibrous polypi. This form of nasal polypus is troublesome to remove, as owing to the cut vessels not being able to contract properly in the dense fibrous tissues it is difficult to control hæmorrhage. Sometimes hard fibromata also develop in the intermuscular septa on the nerve as "false neuromata." In the mammary glands and prostate, fibroma develops in association with gland tissue in the connective tissue stroma of these glandular organs. It may also develop underneath the mucous membrane of the tongue as hard, pale, and pedunculated tumour. *Vide* photo plate No. XXXVII. They are very hard and are described as Adeno-fibromata. The fibroid of the uterus is a Myoma or more correctly Leio-myoma, and not a fibroma. In solid organs a fibroma can be easily shelled out, as a distinct capsule is always formed around them, although they grow by compressing the surrounding tissues.

Hard Fibroma.

Epulis Fibrous-polypus of the nose.

False-neuromata.

Breast adeno-fibromata.

### BLASTOMATOIDS.

Blastomatoids.

(i) Molluscum Fibrosum.

The Fibromatosis in connection with excessive hyperplastic reactive fibrosis in the cutaneous fibrous tissue and fibrous sheath of the cutaneous nerves resultig in



FIBROMA OF TONGUE.



the formation of subcutaneous nodules as happens in **molluscum fibrosum** (Von. Recklinghausen's disease) is a blastomatoid condition and is fully described under **blastomatoids**, *vide* Chapter I. of this volume, page 41.

(ii) Keloids—or Cheloid.

(ii) Keloids  
or Cheloid.

Another condition of hyperplastic reactive fibrosis is met with in cutaneous fibrous tissue as a result of excessive production of fibroblasts in association with some form of irritation, injuries, or operation wounds, known as **Keloids**. This form of fibromatosis is also a blastomatoid condition; for a fuller details of which, *vide* Chapter I. of this volume, page 17.

(iii) Thrombosed piles is an instance of blastomatoid condition of fibrous tissue which resembles a fibroma.

**CLINICAL POINTS.**—The most important point in some fibromata is their superficial degeneration exhibiting an inflammatory zone of infiltration ultimately forming into ulceration, which leads to profuse hæmorrhage. The vascular supply as has already been described is very defective in hard fibromata, but dilated veins are present in the capsule and sometimes in the substance of the mass which become the fruitful source of such hæmorrhagic trouble. Clinical points.

The degenerations that a Fibroma is subject to undergo are, (a) Auto-digestion or necrobiosis; more frequently occurs in fibromyomata than in pure fibroid type of the tumours, perhaps as a result of mild infection; (b) Mucinoid; and (c) Myxomatous degenerations; (d) Ulceration and Suppuration; (e) Calcification. Degenerations.

**CLINICAL SITUATIONS.**—The tissues and incidence of fibromata have already been described, but it must be remembered that in the majority of instances these tumours and blastomatoids are expected in the following situations:— Clinical situations.

- (i) In the skin and subcutaneous tissues, including—

- (a) Von Recklinghausen's disease.  
(Molluscum fibrosum).
- (b) Plexiform Neuromata.
- (c) Keloids.
- (ii) In the submucous tissues—all Polypi.
- (iii) Muscles and fascia.
- (iv) Around periosteum of bones resembling sarcoma.
- (v) In relation to the connective stroma of glands, *e.g.*, breast and prostate.

A FIBROMATOSIS as has already been described is a fibrous tissue overgrowth as happens in an indurated and thrombosed piles. It is closely allied to Fibromata although differs in some important particulars for the details of which *vide* Blastomatoids. In the alimentary canal, kidney, prostate gland, the breast, localized area of tumour-like nodules consisting of fibrous tissue growths are found which are not encapsulated. They gradually merge off into the surrounding tissues. These Fibromatosis are frequently multiple, are of slow growth and often contain gland structures. Sarcomatous change may occur in them although such a condition is rare.

#### Diagnosis.

DIAGNOSIS of real fibroma is often impossible unless a section is examined under microscope; and it must be remembered that the main bulk of other blastomata may consist of hard or soft fibrous tissue. In some cases as in adeno-fibromata the glandular structure may predominate. In others again it is impossible to distinguish it from Sarcoma. In the latter conditions it is the clinical features which help us in coming to a real diagnosis.

#### Treatment.

TREATMENT,—is complete extirpation. The details of the operation vary according to the region involved and are described in the Regional Surgery. *Vide* Volume VI.

## III. CHONDROMA.

## III. Chondroma.

Chondromata are cartilaginous tumours which grow in connection with either cartilage or bone and sometimes in other tissues even where cartilage is normally absent, such as in glands, muscles, tendons, connective tissues, etc. Synovial fringes also afford instances of supplying something like scaffolding for the development of cartilaginous tumours; but these are fibrous than cartilaginous in nature and are blastomatoids. A chondroma may be composed entirely or largely of cartilage.

**APPEARANCE.**—Chondromata are rounded or nearly spherical growths usually of small size. Sometimes they become very large. They are frequently multiple but there is a solitary type also. Their surface is irregular or nodular looking like Bengal custard apple called “ata.” The especial pathological significance of this nodular character is that the line of junction of the adjoining nodules represents the place where the individual nodules are connected together by connective tissue containing blood vessels. These blood vessels run between the series of lobules of semi-transparent avascular hyaline cartilage; the whole mass being enclosed in a more or less definite fibrous capsule.

In **consistence** chondromata are hard and generally encapsuled tumours.

On **section** they present the ordinary appearance of hyaline cartilage, and sometimes in some large tumours cystic cavities containing gelatinous material or isolated softened foci may be seen. Although the cut surface of a chondroma exhibits well-formed hyaline cartilage, the surface is not uniform as it is traversed by fibrous septa carrying blood vessels which divide them into a series of separate lobules forming the nodules, described above. The cut surface looks white or bluish-grey and translucent. Very rarely fibro-cartilage may be detected in some tumours in the neck which may be accessory auricles developing in connection with the branchial clefts.

Microscopical appearance.

Microscopically, the tumour is found to consist of lobules of cartilage separated by intersecting bands of fibrous tissue which contain blood vessels. The cartilage cells occur in spaces and may be rounded or stellate, they thus differ from those met with on the articular surfaces; that is to say they are less regular in shape which is varied, and more numerous than in the normal cartilage, and are irregularly arranged. In some instances the cells may exhibit a branched character and may not have distinct capsules. This feature is more marked in the growing parts of a mass. Sometimes the matrix is found to be distinctly fibrillated like that of fibrocartilage.

Points of difference from normal cartilage.

Peculiar characteristics of Cartilage Tumours.

Cartilaginous tumours have some especial peculiarities of their own which are not found in other non-malignant tumours, *viz.*, (1) Occurrence of Metastases in chondromata, a feature impossible in a tumour if it is non-malignant in nature. (2) Development of Chondroma at peculiar sites, perhaps they are teratomata, or may be due to misplacement of embryonic remnants. (3) Development of Chondroma inside bone perhaps due to misplaced islets of cartilage cells. (4) Unossified remnants of cartilage giving rise to cartilaginous tumour in the bone. (5) They are very prone to become malignant, and are most frequently seen in malignant forms in young adults.

Clinical varieties.

### CLINICAL TYPES.—

Clinically three types of Chondromata are met with, *viz.* :—

(1) **Ecchondroma.**  
Solitary Type.

(1) **ECCHONDROMA.**—These are **solitary** simple out-growths from an islet of cartilage on the surface of the osteum of a long bone situated near one or other end, or in the pelvis, or in the costal cartilage, or in other situations where cartilage is normally present, such as nasal septum, larynx, etc. This **solitary** type of Ecchondroma has no apparent connection with the epiphysial cartilage, and has nothing to do with its development, or

growth. Ecchondroma grows as a hard lobulated mass which may attain any size sometimes as big as a football. The mass is painless and remains attached to the bone. It is the mechanical effects of its size and weight, which produce pain, atrophy of surrounding structures, or obstruction to movement of the limb. This type very frequently becomes **sarcomatous**, when it invades the surrounding tissues and the medullary cavities. Under X-rays, the tumour exhibits islets of clear areas unless calcified.

(2) **ENCHONDROMATA**.—These are always **multiple**. They grow in the short bones of the hands of young persons; less commonly in the bones of the feet. The tumour arises at first in the **cancellous** tissue **near** the epiphysial cartilage in the case of the phalanges and metacarpal bones and cause a fusiform expansion of the finger. The expansion is of diffuse nature and these chondromata from the smaller bones, occur in young people as stated above before ossification is completed. The metacarpals and phalanges are most commonly affected. The actual focus of the growth is the interior close to the epiphysial cartilage. The growth results in exhaustion of the girth of the bone. The young hand gives a clinical picture similar to a gouty old hand. Deformity follows which may affect subsequent growth of the hand.

(2) Enchondroma  
Multiple  
Variety.

(3) **TERATOMATA**.—The next groups of cartilage tumours are those which arise in the regions where cartilage is normally absent but grow in connection with other tissue, *e.g.*, parotid gland, ovaries, testicles, mamma, bursa, etc. These are in reality Teratomata; or mixed growths where the cartilage-element may dominate the picture.

(3) Chondroma of  
Teratomatous  
nature.

**BLASTOMATOIDS**.—Clinically blastomatoids met with in relation to cartilaginous tumour are:—

Blastomatoids.

(i) **ECCHONDROSES**.—These occur in connection with osteo-arthritis, *vide* next volume.

Ecchondroses.

(ii) "LIPPING" along the free edges of the articular cartilages.

(iii) FRINGES (synovial) where cartilage cells form.

It may be summarized that :—

(a) The majority of chondromata arise in connection with the epiphysial junction of the long bones. They are most common in childhood or adolescence, and sometimes several members of the same family may be found to be affected ; and either a history of rickets or injury is available in almost all cases, which indicates that the tumour probably arises in the **arrested** islands of cartilage which have been separated during development of the bone by an attack of rickets or injury.

(b) In some instances the cause of the growth is due to developmental **error**, as from Meckel's cartilage.

(c) Some are examples of **rest**, as in the case of chondroma of the breast. It is possible that the cells are derived from the developing body wall.

(d) Some are clear examples of **metaplasia** occurring in the connective tissue of the organ.

(e) Most of the growths of this nature at places where cartilage is normally absent are **teratomas**.

**DEGENERATIONS.**—Chondroma may undergo the following degenerations which are not frequent, *viz.* :—

(i) True ossification.

(ii) Calcification.

(iii) Mucinoid or myxomatous degeneration, and softening.

**Diagnosis.**

**DIAGNOSIS.**—Diagnosis is not difficult under X-rays, but confusion may arise with the following conditions, *viz.* :—

(i) Callus formation around fractures.

(ii) Chondroma with formation of bursa after separation of epiphysis.

(iii) Blastomatoids of cartilaginous tumours.

(iv) Blastomatoid Giant-celled tumour.

(v) Tuberculous Dactylitis, with the Multiple Enchondromata.

(vi) Periosteal Sarcoma.

(vii) Multiple secondary Sarcomata.

**TREATMENT.**—In the case of chondromata of the Treatment. smaller bones which are usually seen in young people they may be exposed by incising the capsule and then removed by thorough scooping out of the cartilaginous tissue. This may affect the development of the bone inducing subsequent deformity. In the cases where extension has gone too far, amputation is the only method which saves the life of the patient.

In the cases of chondroma of the long bones in a very early condition it is possible to shell it out and thoroughly remove it. This course may not be possible in a tumour of a large size which has invaded other structures. If they are involved with epiphysial cartilage, which is not generally the case, in enchondroma deformity may result. Recurrence is not uncommon due to leaving behind a peripheral lobule in the bone.

Amputation of the limb is usually called for and is inevitable in most of the cases, as if it is not promptly undertaken, sarcomatous changes may occur.

**CHORDOMATA.**—These tumours arise from the Chordomata. remnants of the notochordal tissue. Although majority of them appear to be malignant they grow slowly and seldom disseminate. Those arising in the cranium become serious from their position.

Chordomata are usually situated at the spheno-occipital synchondrosis at the base of the skull, or in the sacro-coccygeal region.

Microscopically, the structure is alveolar and resembles a chondroma excepting that it is a little more cellular. The spaces show a marked tendency to undergo early mucinoid degeneration.

## IV. Osteomata.

## IV. OSTEOMATA.

Tumours composed mainly of bone or calcareous tissue arising in connection with bones entirely limited to the skeletal system are called **osteoma**. In some particular circumstances they may arise in the soft parts.

Different forms of Calcareous tissues.

## DIFFERENT FORMS OF CALCAREOUS TISSUES:—

Osteogenesis.

(a) **OSTEOGENESIS**.—Physiologically bones are divided into two kinds according to their density. Similarly bone tumours are divided into two kinds, *viz.*, **spongy** or cancellous variety, and compact variety sometimes described as **ivory**. We know the process which are at work in these physiological bone formation and we describe such process as Ossification.

Blastomatoids.

(b) **BLASTOMATOIDS**.—Calcareous tumour formation is an instance of another type of bone-formation which is so similar in character to inflammatory hyperplasia that it is often difficult to distinguish these blastomatoid processes from actual blastomata. *Vide* page 22.

Osteoid and Hyperplastic processes.

(c) **OSTEOID and OSTEOLASTIC processes**.—

Callus-formation after fracture, calcification, hyperostoses, are processes of bone-formation which must also be distinguished from Osteomata. A callus is a form of granulation tissue. Osteoblastic granulation during the process of conversion into true bone or osteogenetic tissue is called callus, (*Vide* volume I, part II, and Volume IV). Callus is really a process of regeneration of bone. Hyperostosis is a purely inflammatory process by which diffuse over growths of bone take place under some irritation inducing *infiltration* between the periosteum and the cortex. Occasionally, as described in the chapter on osteomyelitis, bones of the calvarium, or as a matter of fact in certain circumstances all the cranial and facial bones, overgrow enormously by hyperostosis.

(d) METAPLASTIC process.—

Metaplastic process.

New bones are sometimes developed inside the substance of muscles and tendons by metaplasia under the action of constant irritation, *e.g.*, “rider’s bone,” formed in the tendon of the adductor longus of professional riders.

So that we find that the different pathological growths do not differ so far as the histological features of the calcareous tissues or cells constituting these different growths are concerned, but the trouble arises in connection with the accurate knowledge of the **processes** which have been at work to produce or bring forth any particular result. We shall see later on that very few bony growths are real blastomata. *Vide* page 22.

Trouble is to know the different process of bone formation, not their histological character which is always the same.

**OSTEOMATA** may be :—

Osteomata.

(i) **Cancellous Osteomata**, that is, those which are associated with the cartilage bones of the trunk, extremities and base of the cranium having a porous or cancellous consistency.

(ii) **Compact Osteomata or Ivory Osteomata**, or those which are associated with the membrane bones of the cranial vault and face.

It may be noted that both the varieties possess all the histological features of bone, *viz.*, a calcareous matrix, typical osteoblastic cells with processes, and Haversian canals.

(i) **CANCELLOUS Osteomata** are composed of cancellous tissue and often contain cartilage, and their structure is similar to the bone ends which constitute the articular ends of the long bones. They grow near the articular ends of the long bones, or in the vicinity of the epiphysial cartilage of flat bones, and therefore occur almost invariably in young people at puberty or just before it. They usually develop from some isolated portion of the epiphysial cartilage which has become separated from its original connection by the pathological action of an attack of rickets or injury, and then get ossified; and it may be especially noted and remembered that these tumours are

(i) Cancellous Osteomata.

This type is associated with growth and development.

common at the remote ends of the nutrient arteries. Such an island, if placed near the end or surface of the bone, develops into a neoplasm composed mainly of cancellous bone covered or capped **always** by a layer of hyaline cartilage. This is the type associated with growth and development. Many Cancellous Osteomata are really ossifying enchondromata. Or they are sometimes described as **cartilaginous exostoses**.

It is perhaps a Blastomatoid process.

In **form** an exostosis is sessile or pedunculated. In **size** they grow generally to that of a walnut or sometimes they may attain a large dimension, in which circumstance considerable deformity of the part or limb occurs. Since it is a growth at the growing age and is associated with development of the subject the neoplasm ceases to grow further also when the subject attains maturity, or the normal time of fusion of the epiphysis approaches. It may still then remain attached to the epiphysial line, or separate from the epiphysis, when in time the growth of the tumour ceases and the cartilage cap disappears ; a circumstance which is an undoubted evidence that it is a blastomatoid and not a blastoma. The variety called Subungual Exostosis is a blastomatoid and not a blastoma. *Vide* page 22.

Single or multiple.

Cancellous Osteoma are commonly seen on the lower and inner side of the femur near the adductor tubercle, upper end of the tibia, or humerus ; or may grow at any articular end of a long bone. They may remain capped with a **bursal** sac, to be described.

**Exostosis and enostosis.**

Cancellous Osteoma may be Single or Multiple, and as cartilaginous tumours are described as **Ecchondroma** when they arise from underneath the periosteum, or **Enchondroma** when they start from interior of the bone from cancellous tissue, similarly Osteoma is described as **exostosis** when the tumour grows from the outer surface of the bone which is often distinctly pedunculated ; or as **enostosis** when the growth arises in the interior of the long bones and project into the medullary cavity. The former called **Exostosis** is more common. Sometimes

when Osteomas are found embedded among the muscles adjacent to a bone from which however they may be quite distinct, they are described as **parosteal**, Exostoses as they are supposed to be derived from groups of osteogenetic osteoblasts underneath the periosteum which by some means or other such as injury or fracture have become detached and, being separated, are transferred from their normal situation into the substance of the muscles where they continue to proliferate. **Parosteal Exostosis.**

**SINGLE EXOSTOSIS.**—Instances of Single Osteoma or Exostosis are most commonly seen at the inner condyle of the femur and it is the most common variety of Osteoma met with. As described above it grows close to the adductor tubercle and gives great discomfort when riding. But it must be remembered that this is not the type called "rider's bone." **Exostosis at the lower end of femur.**

Single Osteoma at the upper end of the tibia develops on the inner side of the bone. It may sometimes hitch off any tendon which is inserted at or near its neck, and cause painful locking of the knee. **Upper end of tibia.**

Similar Osteoma may develop at the upper end of the humerus after an injury or separation of epiphysis. A bursa may also form on the growth, the cystic character of which may obscure the diagnosis of the osteoma. **Upper end of Humerus.**

**EXOSTOSIS BURSATA.**—Occasionally as described above an adventitious bursa is formed over the prominent part of a single Osteoma where it is subject to constant friction or pressure or both. This form of bursa may be described as Exostosis Bursata. It may not be possible to diagnose the existence of such an Osteoma till accidentally a cystic growth is felt on the top of a tumour, the cyst being formed by the effusion of synovial serum into the sac of an adventitious bursa developed on the surface of the hyaline cartilage cap of the Osteoma as a result of constant friction or pressure. When these bursæ develop in the vicinity of the joint, *e.g.*, the shoulder as stated above the sac may establish communication with **Exostosis Bursata.**

the joint cavity. This is also described as **Osteoma Bursata**.

Multiple.  
Inherited.

**MULTIPLE EXOSTOSES.**—Multiple exostoses are more frequently met with when they develop in large numbers; sometimes as many as hundreds have been reported to occur on a single person. These are often inherited, and may be seen among several members of the same family. Pathologically, they are similar to the single exostoses, only affecting many bone ends at a time.

(ii) Compact or Ivory Exostoses. They develop in the cranium.

**(ii) COMPACT OR IVORY EXOSTOSES.**—Are composed of masses of very dense compact bone. They differ from normal compact tissue in having a fewer Haversian spaces and canaliculi.

Ivory exostoses are also common amongst young people; but unlike the cancellous exostoses they occur in middle age also.

Sites.

They develop in the cranium in connection with the membrane-bones of the vault of the skull where they most frequently grow, (a) on the inner, projecting into the cranial cavity; or (b) outer, tables of the bones, when they form a hard rounded sessile mass, especially in connection with the orbit, frontal sinus, antrum, auditory meatus, etc. They are also found on the bones of the face, ilium, and scapulæ.

Pathological characteristics.

**PATHOLOGICAL CHARACTERISTICS.**—Ivory Osteomata are composed of compact osseous tissue covered by periosteum with fewer Haversian spaces and canaliculi. Such composition makes the growth *very* dense so much so that sometimes they cannot be removed by forceps or, saws, and it is for this reason they are described as **ivory** exostoses. The Haversian systems are more irregular and are usually arranged at right angles to those of the normal bones. They are generally single and small and are usually lobulated. The size do not often grow bigger than a large betel nut, and as has been stated before they are chiefly found in connection with the bones of the skull.

The direct effects of the growth are more mechanical in nature than pathological ; and chiefly by pressure and obstruction these growths, *e.g.*, near the orbit may cause to interfere with the eye sight or to destroy or displace the globe ; or invade the antrum of Highmore or various sinuses ; or a similar growth situated in the frontal sinus or on the under surface of the skull may produce grave symptoms of irritation such as Jacksonian or true epileptiform fits, and cerebral compression of the brain ; or compression of the membranes ; or the principal cranial nerves. Otherwise they may not be detected or, as they may not produce any symptoms, effect any immediate fatal result.

In some cases degenerative changes such as necrosis may occur, in which circumstance the growth may be sloughed out and end in spontaneous recovery.

To help our memory to remember the different varieties of Osteoma they are set out in a Table, *vide* next page.

The other so called Exostoses such as Subungual Exostosis, or Dental Exostosis are not Blastomata.

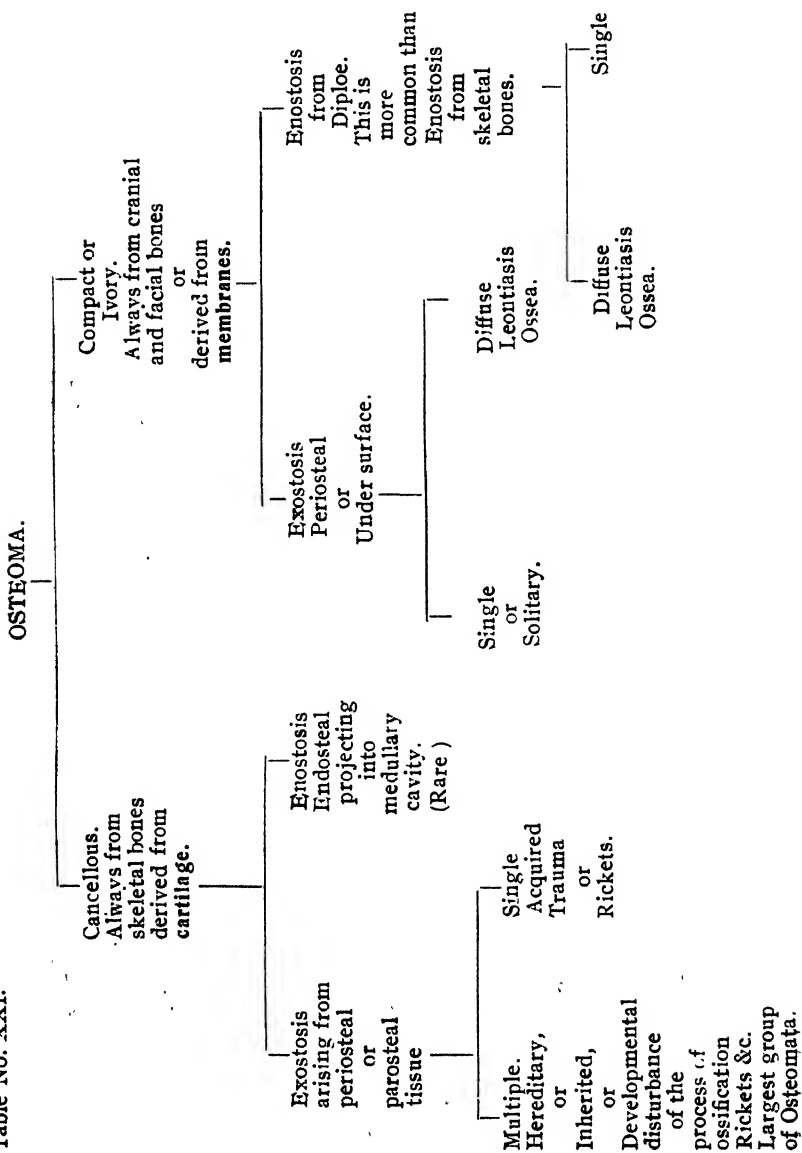
TREATMENT.—Treatment of Exostoses consists Treatment. entirely of extirpation. It must be borne in mind that Exostoses cease to grow at the time when the epiphysis joins the shaft and this point should be considered in connection with the advisability of immediate operation, unless necessitated by pain or other urgent symptoms.

A growth in the limb could be removed by chiselling it away without much difficulty ; but it becomes a very serious matter if it were an operation on a growth of compact variety developing in the calvarium.

In removing an Osteoma especial attention should be given to the total extirpation of the cartilaginous tissue covering them, as any portion of such tissues left behind will surely lead to recurrence. It may otherwise be easily chiselled or sawn off if growing on the extremities.

In the calvarium it may not be possible to remove the growth by chiselling or sawing. In such instances a burr driven by electricity may be used to divide their attach-

Table No. XXI.



ments. In no case Ivory Exostoses should be interfered with unless the symptoms are of pressing nature and is imperative.

DIAGNOSIS.—Diagnosis rests on differentiating it from the following conditions:—

(1) Inflammatory growths, *e.g.*, excessive callus, Brodie's disease, &c. Osteo-arthritis, Charcot's disease.

(2) Blastomatoids *e.g.*, Giant-celled tumours, Myositis-Ossificans.

(3) Blastomata-Nonmalignant,—Ossifying Chondromata, Leontiasis Ossea.

(4) Blastomata-Malignant,—Sarcomata.

## V. MYXOMATA.

V. Myxomata.

The Myxomata as the name implies are *supposed* to be tumours formed of mucous like or mucoïd tissue. Myxomatous tissue is not a normal tissue of the adult but in the foetus it constitutes the Wharton's jelly in the umbilical cord.

APPEARANCE.—True Myxomata are described as rounded, lobulated, elastic, semi-gelatinous, translucent tumours, resembling boiled sago or thick white phlegm, which may occur in any connective tissue, and are very liable to become malignant. They are encapsuled and grow slowly. On section they exude mucinous fluid.

Microscopically, myxoma appears to constitute of connective tissue cells surrounded by and separated from each other by mucoïd substance similar in character to Wharton's jelly in the umbilical cord. The cells have long branched processes and are polygonal in shape. These processes interlace with processes from adjacent cells. The nuclei are dark staining, and exhibit a fair amount of cytoplasm in them. The inter-cellular myxomatous substance is translucent and homogeneous in appearance, and sometimes a few wandering connective tissue cells and leucocytes are detected at some areas. Blood vessels traverse the substance of the tumour.

Microscopical features.

Consistence.

The consistence and density of the tumour varies according to the proportion and amount of intercellular substance.

True Myxoma is perhaps unknown? or very rare.

It must be remembered that most of the so called Myxomata are really not Myxomata at all, or have nothing to do with Myxomata, *e.g.* :—

(i) Œdematous Fibromata or Myomata with hyaline or mucoid degeneration are often described as Myxofibroma.

(ii) The mucous polypi of the mucous membrane in the nose consist of a mass of delicate fibrous granulation tissue with few cells, imbibed with œdema fluid in its inter-spaces, and covered with ciliated epithelium and they are neither Myxomata as formerly described, nor they have anything to do with Myxoma.

(iii) Similarly, in the ear described as aural polyp, myxomatous masses are met with which are also of the same nature as the above.

(iv) Inflammatory masses of œdematous granulation tissue as are met with in connection with the ethmoid and petrous bone are sometimes wrongly described as Myxoma.

(v) Some growths, *e.g.*, in the stomach which are distinctly colloidal in nature and although described as Myxomatous are really Colloid Carcinoma.

(vi) Some Gliomas in the central nervous system often become so semi-translucent that they are described as Myxoma. Such tumours are in reality degenerated glioma.

(vii) Myxo-Sarcoma is merely a degeneration and not a true neoplasm.

Myxoma is perhaps a degeneration.

It is therefore often suggested that Myxoma is mucinous degeneration, and not a neoplasm.

Shall we deny their existence?

We cannot all the same deny their existence in "toto," as recognized authorities have described pure Myxomata. It is described that they do occur in subcuta-

neous and subserous tissues, in bones, muscles, and endocardium.

As has already been stated Myxomata soon assume malignant properties. Quick tendency to Malignancy.

TREATMENT is complete extirpation. They must be thoroughly eradicated as otherwise chance of their turning malignant or recurrence remains always great. Treatment.

## VI. MYELOMA. OR GIANT-CELLED TUMOURS.

VI. Myeloma.

As has already been described, Myelomata are not Blastomata but are Blastomatoids, and they are described in the first chapter. They cannot hold any seat amongst true tumours. *Vide* p. 23.

## VII. LYMPHOMA.

VII. Lymphoma.

LYMPHOMA means a blastoma composed of lymphoid tissue ; which may be a reactive hyperplastic condition of any established lymphatic glands ; or any fresh nodes of a typical structure making a new appearance. It is doubtful if there can be any neoplasm composed really of lymphoid element. Commonly by Lymphoma we understand all neoplasms having a lymphoid structure. Such lymphoid tissue may be, (a) accumulation of a mass of lymphoid tissue in the spleen or other organs in leukæmia, (b) there may be lymphoid hyperplasia, as in lymph-adenoma or Hodgkin's disease, or (c) in association with other growths, *e.g.*, Lympho-sarcoma. Diagnosis of a real blastoma composed of lymphoid tissue only is a matter of difficulty ; but blastomatoid conditions of lymphoid tissue are of greater interest.

SIZE.—Lymphomata vary in size. They may be single or multiple. Solitary types are rare but when they develop as a single tumour they do not attain a size bigger than a hen's egg. Size.

Multiple ones may form a mass of considerable size and in the neck they may be mistaken for tuberculous glands.

Site.

**SITE.**—Its most common situation is the glands of the neck. It may affect one or more glands at a time.

Character.

**CHARACTER.**—A few examples of this form of growth are encapsuled, and they like other non-malignant neoplasms do not show any tendency to infiltrate in the neighbouring tissues. They do not show any signs of inflammatory indurations or adhesions except those of blastomatoid nature which may involve the neighbouring structures.

On section, they present the appearance of a normal lymphatic gland except for their unusually bigger size. Microscopically, they resemble typical lymphoid tissue, and even then they exhibit the characters more of reactive hyperplastic changes than of Neoplasms. It must be always considered that lymphoid tissues are very widely distributed throughout the body and they readily undergo hyperplasia in response to any irritation or infection.

Hodgkin's disease.

The Lymph-adenoma known as Hodgkin's disease is of course not a tumour of the lymphatic glands. This form of growth is characterized by their progressive enlargement of the lymphoid tissue mainly of the glands of the neck, and also of spleen liver and other organs. The extension takes place by contiguity. The affection presents a picture seen in reactive hyperplasia, or in infective hypertrophy. *Vide* photo plate No. XXXIV.

Lympho-Sarcoma.

**LYMPHO-SARCOMA.**—The term signifies different meanings ; but it is generally restricted to malignant tumours which have a structure resembling lymphadenoid tissue. Some of course closely resemble a small-round-celled sarcoma with the exception of the stroma being in abundance. Their nature of malignancy is evidenced more by clinical significance than by their histological characters ; as they soon destroy and invade surrounding structures. For further details *vide* p. 247.

For the differential diagnosis and details, *vide* Volume V.

### VIII. MYOMATA.

VIII. Myomata.

A Myoma is a neoplasm consisting of muscle tissue. Muscle fibres are physiologically of two kinds namely striped and unstriped. Similarly blastomata arising from them are also of two kinds, *viz.* :—

#### (i) RHABDO-MYOMA.

(i) Rhabdo-Myoma.  
Very rare neoplastic fibres never attain the pathological standard.

Tumours composed of striped muscle fibres are called Rhabdomyoma, and a glance at the Table of classification after Adami would show that this form is a **mesothelial hyloma**. Clinically, Rhabdomyoma is exceedingly rare. In fact the myomatous tissue never attains its full development of striated muscle in a neoplasm and so also mature striated muscle cannot give rise to blastomata. The large number of cells present in a Rhabdomyoma are of embryonic type and for this reason it is often described as sarcomatous in nature. Rhabdomyoma probably arises from embryonic residues from foetal rests, and the normal structure of striated muscle is never fully reproduced in a Rhabdomyoma. These neoplasms are generally composed of a vascular connective tissue frame work containing a large number of round and spindle cells with a few more or less ill-developed striated fibres of muscle cells which are always irregular both in size and shape. The skeletal muscles are very seldom affected by this type of tumours; but a few instances of this kind are seen. Generally these are found to arise in the heart, uterus, œsophagus, and kidney, the last seems to be the commonest seat. Testes and ovaries are also sometimes affected. Instances of Rhabdo-sarcomata and mixed tumours especially in the form of renal sarcomata of infants called Wilm's Tumour are found. *Vide* p. 253.

(ii) Fibro-Myoma or Leiomyoma or Fibroids. Whereas the former type is rare, this type is very common.

## (ii) LEIOMYOMA.

Tumours composed of unstriped muscle fibres are on the other hand very common and their incidence rank practically highest. They are called Leiomyomata or Fibromyomata. The most familiar instance of Leiomyoma is the uterine Fibroid. It is of course described as "fibro-myoma" when there is an abundance of normal interstitial connective tissue.

Adami's classification will show that Leiomyoma is of **mesenchymal** origin.

It should be remembered that although Leiomyoma is a very commonly met with tumour, neoplasm which can be histologically called pure Leiomyoma is rare. Most of the tumours classed under such description are Fibromyoma, that is to say contain abundant fibrous tissue.

Appearance.

**APPEARANCE.**—The uterine Fibro-myoma is an encapsulated, large, hard, sometimes pedunculated or sessile may be rounded or lobulated, dry, firm, and an elastic tumour. It may be single or multiple, and sometimes it attains a very large dimension and size.

In the alimentary canal, bladder, and below the capsule of the kidney they grow only of very small size.

Alimentary canal  
Bladder  
and kidney.

In the intestines a tumour tend to become polypoidal which may produce mechanical obstruction or interference of function.

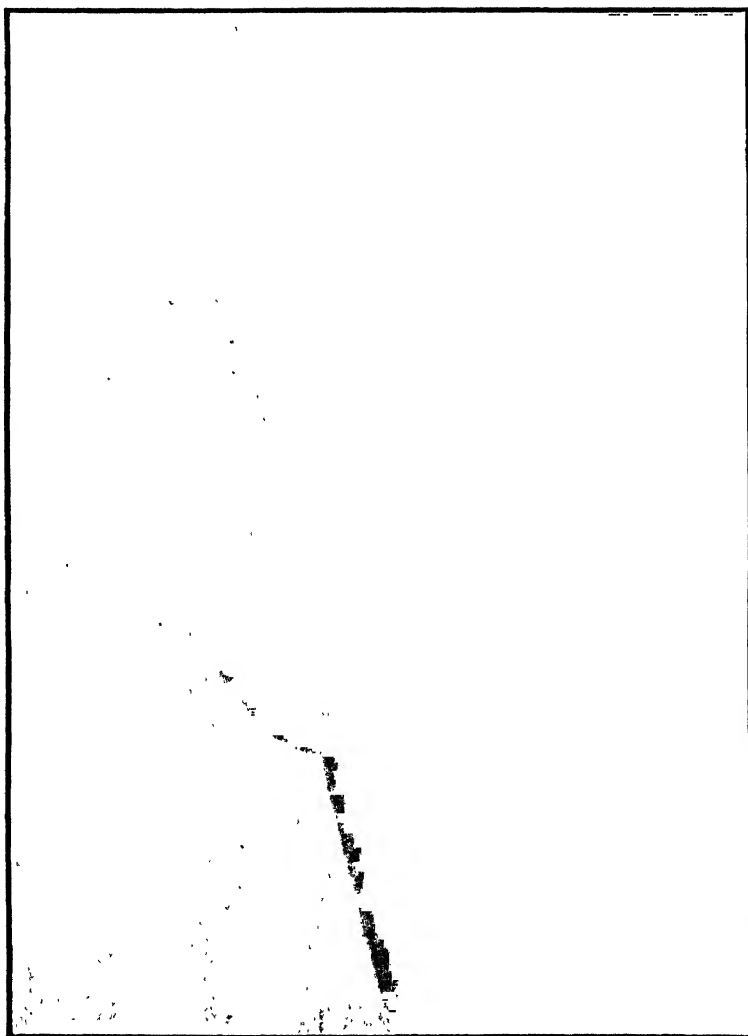
Skin.

In the skin the growths appear like little nodules only.

Naked eye appearance on section

Leiomyoma has always a distinct capsule. In the uterine "fibroids" the cut surface presents a paler look than the uterine walls because the growth is less vascular than the normal wall. The surface resembles that of a fibroma. It has a fibrous look but the fibres are more regularly arranged and they appear to be more elastic than those of fibromata. The neoplasms themselves are not very vascular but vessels of large size develop in the capsule. The blood vessels have distinct and definite





FIBRO-LEIOMYOMA OF SKIN.  
On The Thigh.  
(Highly Magnified).

walls, a very important point and a distinguishing feature from sarcoma.

Microscopically, Leiomyoma are seen to consist of little bundles of spindle-shaped cells which are long and fusiform and contain rod-like nucleus. Bundles of these cells are grouped together into fasciculi but the peculiar distinguishing feature from fibroma is the arrangement of these bundles. They are not arranged like "whorls" and the characteristic wavy tendinous fibrillæ of a fibroma are absent. In Myoma these fibres are more or less regularly arranged. Yet it is often difficult microscopically to distinguish a myoma from a fibroma. On the other hand in some instances it becomes very difficult to distinguish it from Fibro-sarcoma, as tissues similar to those of fibromyoma may occur in sarcoma. It should always be remembered that it is sometimes very difficult to distinguish between, (a) the unstriped muscle cells, (b) fibrous tissue cells, and (c) cells of spindle-celled sarcoma. In sarcoma, the growing edge is more embryonic in character, but in myoma, the cells and the structure are of the same nature throughout; moreover other types of cells and tissues may be marked in sarcoma. In sarcoma the blood vessels are mere interstices and clefts, but in myoma they consist of well formed walls. Van Gieson's stain is a very useful method to clear our difficulty as it helps us practically to differentiate fibromyoma from other kinds of tumour by exhibiting the muscle in yellow and the fibrous tissues in pink.

**SITES.**—Leiomyoma or Fibromyoma develops usually where the unstriped muscle fibres physiologically exist. These being the uterus, ovaries, Fallopian tubes, the prostate, the alimentary canals, from the middle of the œsophagus to the internal sphincter ani, beneath the capsule of the kidney, the bladder and lastly the skin. *Vide* photo plate No. XXXVIII. But the uterus is the commonest site for fibromyoma in women especially described by gynecologists as **fibroid**; and the prostate is similarly the

site affected in men. The next site of distribution is the stomach; the œsophagus and the intestines follow the last closely.

The  
uterine  
Fibro-  
myoma.

Above all in the uterus Fibro-myomata assume such notable proportions as to warrant us to bring them into a class by themselves.

The uterine Fibromyomas are classified as follows:—

(a) Intra-mural, that is, when they develop from the walls of the uterus.

(b) Sub-mucous, that is, when they develop from beneath the mucous coats and hang like polypi in the cavity of the uterus.

(c) Sub-peritoneal, when they encroach upon the peritoneal cavity. These develop from underneath the serous coat of the peritoneum and are sometimes described as of sub-serous type. They usually grow from the fundus.

The last variety may be sessile, or pedunculated having a pedicle or a stalk.

The  
Secondary  
changes.

The degenerations and secondary changes that may occur in an uterine Myoma are the following:—

(i) Red necrosis resulting from interference of circulation, due to usually the stalk being twisted. This condition is sometimes described as **red degeneration**.

(ii) Calcification of the dead parts.

(iii) Ulceration with profuse hæmorrhage; sometimes turning it to malignancy.

(iv) Mucinoid degeneration and softening; as well as hyaline change may also take place.

(v) May become sarcomatous.

(vi) Sometimes partial calcification may result in some muscle tissues, the part being encapsuled in the calcareous trabeculæ.

Treatment.

TREATMENT is complete extirpation. The details of the operation varies according to the sites for which, *vide* Regional Surgery.

# LEIOMYOMA CUTIS.

This is a non-malignant growth composed of smooth muscle fibres growing on the skin. We should remember that in the cutis vera the hairs are controlled by involuntary muscle fibres, called Arrectores Pilorum. The disease is a very rare one. The growths consist of reddish nodules which are sometimes bluish or yellowish in colour, ranging from a pin's head to that of a split pea in size. They generally grow on the extensor surface of the forearm, wrist and sometimes also on the cheek. Several hundreds in number may grow at a time. They are tender upon pressure in the recent condition, but become painful when they are old. Cases are reported to have suffered from this form of tumours for fifteen and even twenty years or more without showing signs of malignancy.

Leiomyoma cutis.

Only microscopical examination could render the diagnosis certain.

TREATMENT.—X-rays, or radium are of no avail. Cauterization with pure Trichlor-acetic acid may prove successful.

## IX ANGEIOMA.

It must be distinctly understood that growth of tumour of the blood vessel may take place in connection with :—

(i) Proliferation of simple connective tissue of the vessels, in which case the neoplasm will be a **histioma**, that is to say, hylomata of non-malignant nature; or on the other hand;

(i) Non-malignant histioma.

(ii) The growth may be of the lining endothelium or the lining cells of blood vessels, in which case the tumour as a rule becomes a **cytoma**, which means a malignant form of the growth in relation to endotheliomata, which are lepidomata we are just going to describe.

(ii) Malignant Cytoma.

The majority of vascular histiomas described as Angeiomas are probably not blastomata at all. They

rather appear to be of the nature of simple dilatation and elongations of pre-existing vessels than true neoplastic growths.

We shall find in the Adami's system of classification that *Angeioma* comes under the heading of *hylomata* of mesenchymal origin. Whereas *endotheliomata* come under *lepidomata* arising from transitional cells passing through a mesoblastic stage and are grouped as endothelial *lepidomata*.

The non-malignant endothelial tumours are not difficult to be recognized, but confusion arises in the malignant forms, because no fixed standards are clearly defined by which the neoplastic proliferation of the endothelial cells can be determined.

When the growth of *endothelioma* takes to the forms of *cytomata*, since their transitional cells pass through a mesoblastic stage as stated above, they may advance further, to assume purely epithelial characters that is to say, become **lepidic**; or revert back to more primitive type of cells and become sarcomatous, or **hylic**.

We shall discuss this point in a greater detail in connection with malignant tumours. So far as *Angeiomata* are concerned, since it is convenient to describe all forms of tumours in connection with vascular system under *endotheliomata* which are lepidic tumours, we describe them under the lepidic tumours accordingly. *Vide* p. 135.

## HYLOMATA OF EPIBLASTIC ORIGIN.

### X. Glioma.

### X. GLIOMA.

Glioma is a neoplasm of the central nervous system, arising from the neuroglia. The brain, and the spinal cord are the sites where Glioma develops generally, of which the great majority occur in the brain. It is epiblastic in origin. But although the neuroglia or spider cells originate from epiblastic tissue they function like

the connective tissue, as they support the nerve cells and nerve fibres in both grey and white matter of the central nervous system, and assume the character of hylic or pulp tissue tumours. Nevertheless, the cells of the new growth may remain distinctly epithelial in type rendering a diagnosis a matter of much difficulty. Chemically neuroglia is very different from connective tissues. It is composed of some horny substance called neuro-keratin which is an insoluble matter similar to keratin found in the surface layers of the epidermis.

Gliomata rarely grow of much large size. They may be single or multiple. The colour closely resembles the brain substance and is often not demarcated by any sharp line, to enable the tumour to be distinguished from the surrounding tissues, there being no capsule formed around it. Sometimes a Glioma is distinctly of greyish or reddish or pinkish colour.

Gliomata are as a rule soft in consistence. They vary in progress and rapidity of growth; a hard and a soft variety being thus recognized; and according to the vascularity, the colour may also vary. The blood vessels are badly supported, and therefore hæmorrhage frequently occurs; and due to this alterations in their vascular supply cystic degenerations are common.

Microscopically, a glioma consists of glia cells. These cells have a round or oval deeply staining nucleus with scanty cytoplasm but provided with many delicate fibrils which branch and interlace with those of their surrounding neighbours. In these are incorporated variable amounts of other round cells with little intercellular net work, or large cells provided with sometimes two or three nuclei, and also variable amounts of nerve fibres and nerve cells. The tumour is not encapsuled.

Gliomata produce no metastases; and secondary growths from a pure Glioma do not occur. They are typically non-malignant, but their position makes the case fatal. Sometimes they undergo a malignant transformation.

Non-malignant Gliomata sometimes occur outside the cerebro-spinal nervous system, *e.g.*, they are sometimes found in the medulla of the supra-renal gland or in connection with the retina; but usually these are malignant, and they give rise to metastases. They are sometimes described as Glio-sarcomata, or Neuro-blastomata.

Other variations.

Sometimes the cells of a glioma resemble epithelial cells much more than glial cells, or may be isolated, bearing a striking resemblance to ganglion cells, or sometimes they may be arranged in solid alveoli. In some instances again they occur as linings to well formed cystoma suggesting the cells to be of the ependyma, and the growth having this last feature is described as **ependymoma**.

Ependymoma.

Gliomatosis.

Our conception of what constitutes a Glioma is now materially changing. A history of some kind of trauma may be obtained from the majority of the cases and it is therefore now believed that the process is a more reactive hyperplastic proliferation of progressive blastomatoid nature, rather than a blastoma. At least many of them are really **gliomatosis** of progressive type there remains to have no doubt. *Vide* p. 43.

Diagnosis.

DIAGNOSIS of brain tumour is difficult and the help of a neurologist should always be sought for.

Treatment.

TREATMENT.—*Vide* Regional Surgery.

## XI. NEUROMATA.

XI. Neuromata.

Neuroma is a tumour arising from **neurone** and it therefore consists of newly formed ganglion cells or nerve cells, and nerve fibres; as a neurone is constituted of both cells and fibres. But true Neuroma is so rare that not more than half a dozen of undisputed cases are yet in record. In any case some distinctive neoplasm of this nature can be recognized, although nerve tissue is very liable to various blastomatoid processes. Conditions which are more involved with the proliferation of fibrous

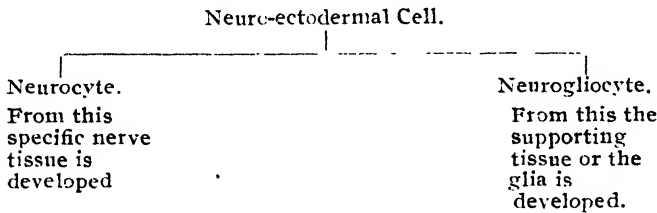
Many are blastomatoids.

tissues either of blastomic process or of blastomatoid process should not be mistaken with the blastomata of the essential constituent of the neurone; as it is the **nerve cell** which is more concerned here although **fibre** exists as a supporting tissue which may also proliferate along with the cell.

Therefore, fibrosis, fibromata, reactive hyperplastic processes of nerve fibres as are met with in *amputation* neuroma have nothing to do with true blastoma described as **Neuroma**. *Vide* p. 42.

During development the nerve tissues and systems undergo many different stages and consequently tumours arising from nerve cells also vary according to the corresponding developmental stages. They therefore contain elements which are not pure but which represent nerve cells in different stages of development.

The whole nervous system is derived from the primitive neuroectodermal cell. This differentiates early as follows:—



Some neurocytes migrate ventrally and form the paraganglia and the sympathetic chain, some of which again form the medulla of the adrenal after penetrating into its cortex. In the adrenal and the other paraganglia there neuroblastic cells differentiate into the ganglion cells and the chromaffine cells later on.

Pathologically therefore, the following varieties of Neuromata are met with, *viz.*:—

- (1) Neuroblastomata.
- (2) Ganglioneuromata.
- (3) Paragangliomata.

Pathological varieties.

### 1. Neuroblastomata.

## 1. NEUROBLASTOMATA.

This variety has the widest distribution. Usually Neuroblastoma arises from the central nervous system, its outgrowths such as the retina and also the sympathetic system. It is most often seen in the adrenal. The tumour is more common than is generally supposed. The tumour is more cellular and is composed of cells having their cytoplasm prolonged at one point into a fibril resembling the axon of the adult nerve cell. The power to form fibrils and their cellularity vary according to the rate of growth. Such forms are often malignant and give rise to metastases. We shall discuss them further under malignant growths.

### 2. Ganglioneuromata.

## 2. GANGLIONEUROMATA.

These varieties are exceedingly rare tumours. They occur in young subjects, generally congenital, often multiple and grow sometimes to considerable dimensions, but are practically always innocent.

Ganglioneuromata are found in connection with the sympathetic ganglia of the thorax, and the abdomen, the medulla of the adrenal, and in the ganglia in the walls of the arteries in the skin. The tumour is composed of ganglion cells in large numbers together with well formed and in some instances even medullated nerve fibres.

### 3. Paraganglioma. Carotid Tumours.

## 3. PARAGANGLIOMATA.

These arise chiefly in the carotid body and adrenal, and are composed of chromaffine cells in various stages of differentiation. They are non-malignant in nature. In some rare instances nerve elements may also be present.

In Teratomata they are found more frequently.

### Clinical varieties.

Clinically, neuromata and all blastomatoids arising in connection with the nerves and their sheaths are described under two broad classes, *viz.* :—

(a) True Neuromata.

A. True Neuromata.

(b) False Neuromata.

B. False Neuromata.

A. True Neuromata as described above are very rare.

B. False Neuromata.—These are blastomatoid growths for the detailed description of which the reader is referred to the previous chapter on **blastomatoids**. Some of these are described as **neuro-fibromata** under Fibroma but strictly speaking they are not blastomata and are neither Fibroma nor Neuroma. *Vide* p. 36.

## B. LEPIDOMATA.

B. Lepidomata.

### I. ENDOTHELIOMATA.

I. Endotheliomata (Non-malignant).

For the purpose of convenient description Endotheliomata may be divided into three main groups; *viz.* :—

- (1) Vascular Endotheliomata.
- (2) Lymphatic Endotheliomata.
- (3) Serous Endotheliomata.

Each of these again may be subdivided according to the particular type of vessels or serous membrane affected which will be described under their main headings.

Non-malignant Endotheliomata of the first two groups that is to say, which arise in connection with blood and lymph vascular system are in practice called by one common name—Angeiomata.

Vascular Endotheliomata and Lymphatic Endotheliomata are practically called by one name Angeiomata with proper prefixes.

Angeioma includes a variety of neoplasms composed of vessels, or in which vascular elements predominate. This is a form of benign growth which appears earliest in life except the dermoids which are really cysts or Teratomas. This form of neoplasm is well-known to all lay people also, and is described by them in various popular terms such as 'birthmarks', portwine stains,

Angeiomata are congenital.

nævi, 'mother's mark', etc. These Angeiomata are either congenital in origin, or they develop soon after birth. They generally involve the skin or mucous membrane, and in some instances the underlying muscles and tissues; and according to the situation and the character of the vessels of which they are formed they present various appearances. They are sometimes acquired and in such instances they develop in the internal organs such as the liver.

Rarely acquired.

Two groups of Angeiomata.

1. Hæmangeioma.
2. Lymphangeioma.

According as the vessel contains blood or lymph the different varieties of Angeiomata are distinguished and classified under their distinct groups described above; such as *hæmangeioma* denoting a *blood-vascular* tumour of Vascular Endotheliomata, and *lymphangeioma* or Lymphatic Endotheliomata denoting *lymph-vascular* tumours. Vascular tumours or aneurysms produced by the dilatation of pre-existing vessels, or overgrowth of pre-existing vessels are not included under this variety of tumours. But the line of clinical demarcation is vague.

1. Vascular Endotheliomata or Hæmangeiomata. Three Types.

### 1. VASCULAR ENDOTHELIOmata OR HÆMANGEIOMATA.

According to the structure of the vessels Hæmageiomata are divided into three types, *viz.* :—

(i) Capillary or simple Nævus,

(i) **THE CAPILLARY ANGEIOMA; OR SIMPLE NÆVUS**, or the generally well-known 'Mother's Mark'. This form is exceedingly common. It is composed of a mass of dilated capillaries held together by a small amount of connective tissue containing fluid blood.

It is generally situated in the skin, although in some cases it also involves the subcutaneous tissues, and muscles.

In this form the tubular form of the individual vessels persists but they are often dilated. The walls of these vessels are usually of more cellular nature than

of the normal capillaries. The endothelial cells are more prominent, and they are supported by little or scanty stroma of connective tissue; in contrast to fairly dense fibrous tissue in the cavernous variety. In some varieties especially in the nodulated types large vessels may be seen ramifying in them.

**APPEARANCE.**—With the naked eye these angioma-  
mata appear as slightly raised flattened mass the surfaces  
of which are sometimes irregular or nodulated or in some  
instances they are markedly elevated or even pedun-  
culated. Appearance,  
shape and  
surface.

In **size** they vary from quite a small tiny patch, of the size of a split pea to an inch, or even two inches in diameter; and in some instances extending a whole limb such as an arm and the neck. Several isolated patches may be seen in the same individual at the same or different sites of the body.

In **colour** they look bright red, purple, reddish or  
violet, according to the relative amount of arterial or  
venous blood contained in them. Colour.

**LOCATION** or site incidence.—These angioma-  
mata are generally found on the head, face, neck, back of the  
mucous membrane, they often become sources of  
dangerous or fatal hæmorrhage. Such instances are  
found inside the nose, bladder or pelvis of the kidney.  
In the last situation it is of gravest clinical importance. Sites.

**CLINICAL FEATURES.**—Angioma-  
mata are usually congenital, and sometimes they appear after birth. Clinical  
Features.

Clinically three varieties of simple or capillary Nævus  
are met with, viz. :— Three  
clinical  
varieties  
of Capil-  
lary Nævus.

(a) Mother's Mark, described above.

(a) Mother's  
Mark or  
simple  
Nævus.

(b) Port-wine stain or Telangiectases and Nævus unius lateris.

(b) Port wine stain or Telangiectases and Nævus Unius Lateris.

(c) Spider Nævus or Nævus Araneus.

(c) Spider Nævus or Nævus Araneus.

The port-wine stains or **Telangiectasis** are more superficial in nature and often extends widely over the back of the upper arms, neck and face. The colour is somewhat dusky. The reason of its not being raised over the surface of the skin is due to its being composed of net works of very fine capillary vessels. In fact the growth is hardly a tumour or a swelling.

Nævus unius lateris.

A condition called **Nævus Unius Lateris** is occasionally met with where a nævoid or nævus-like development is found to extend the whole arm in its long axis, running at the back and external aspect, having a linear distribution, which runs transversely half round the face neck, and the trunk and limited almost exactly by the middle line. *Vide* photo plate No. XXXIX. The skin looks hypertrophied and covered with small papillary excrescences. In some instances the area of the skin affected may look only stained exhibiting purely a vascular manifestation.

**ACQUIRED TELANGIECTASIS OR DE MORGAN'S SPOTS.**—Sometimes in middle aged people port wine spots appear on the back or on the whole trunk as small red spots in large number. They persist for sometime and then disappear. These are possibly the effects of some degenerative process. Microscopically, they reveal the nature of dilated capillaries. Sometimes they appear in women suffering from mammary cancer. This condition induced some observers to propound the theory of their being associated with cancer, but latest observations do not support the condition to have any such inter-alliance.

**THE SPIDER NÆVUS OR NÆVUS ARANEUS** is a form of small angioma which looks like a spider;



NÆVUS.



consisting of a small patch from which radiate a series of fine red lines. It is generally met with on the face of young people. These nævi bleed very easily if irritated, but they are very amenable to treatment by pointed cautery or application of carbonic acid snow.

**PROGRESS AND TERMINATIONS.**—Capillary nævi may remain unchanged throughout life or may in some cases disappear. More commonly they increase in size and when they do so they invade the surrounding tissues. In some instances they remain dormant till the middle age when they suddenly increase in size and extend. They may thus give rise to vascular, or pendulous, or pedunculated, cavernous angioma. If irritated by the application of chemical substances they may ulcerate and sometimes alarming hæmorrhage may be induced.

**TREATMENT** is simple. Application of carbonic acid snow, or a pointed electric cautery with high frequency current would completely cure small nævi. Electrolysis in exposed situations yield satisfactory results. Above all excision is the least troublesome and most successful course to adopt in the majority of the instances.

## (ii) CAVERNOUS ANGEIOMA; AND HÆMANGEIOMA SIMPLEX.

(ii) Cavernous Angioma, and Hæmangioma Simplex.

**HÆMANGEIOMA SIMPLEX.**—A Cavernous Angioma is a typical blastoma only simulating and resembling a cavernous or erectile tissue like corpora cavernosa of the penis. It is constituted of large irregular *spaces lined by endothelium* forming into thin walled cavities in which the arteries often open directly. The cavities thus formed communicate with each other very freely. Unlike that in the capillary types, here in this form the original shape and the tubular form of the constituent vessels are lost and they are converted into irregular spaces. Cavernous Angioma is found chiefly in the liver.

Appearance  
typical  
tumour  
circumscribed or  
diffuse  
shape.

Cavernous Angeioma in the form of nævus very often involves the subcutaneous, submucous or muscular tissues, by merging gradually into the surrounding structures. They form small lobulated growths. They may often be seen in the walls of bursæ at an early stage in their formation. They are sometimes described under a especial name called **Hæmangeioma Simplex**. They are never provided with a capsule, and are not definitely localized. In some cases Hæmangeioma Simplex is associated with superficial capillary nævus of the first group. The fibrous tissues which support the blood spaces are usually fairly dense and they contain few cells; in contrast to scanty stroma of connective tissue in the capillary type.

In **appearance** they exhibit the nature of small typically lobulated tumours, as occur in Hæmangeioma simplex; or a mass which may be definitely circumscribed or diffuse, as is formed in the Angeioma in a liver.

In **shape** they are generally irregular, spherical or pedunculated, or small lobulated growths.

The **surface** is soft to the touch, and on pressure the tumour is compressible like rubber sponge, refilling with blood no sooner the pressure is removed.

In **size** they vary from the size of a small nævus to a more or less prominent tumour.

If it is subcutaneous the **skin** covering the tumour looks bluish in colour; but if the skin is involved it presents a dusky red hue.

On touch no **pulsation** is felt. No bruit could be detected although in some instances of pronounced type both may be present.

**SITES.**—Hæmangeioma Simplex generally occurs on the skin of the face, trunk, the limb or the muscles of the back. In the *liver*, Cavernous Angeiomas are very common. In the latter instances they are formed by the *dilatation of the capillaries between the lobules*; the liver substances gradually disappearing by simple atrophy.

Visceral  
form in  
the liver.

It never forms a large tumour in the liver, but replaces a small portion of the liver substance. In the liver it is the commonest type of new growth.

**CLINICAL FEATURES.**—In visceral angiomas Clinical Features. such as that in the liver it exhibits no characteristic clinical feature and gives rise to no such symptoms as to lead one to its diagnosis. It is usually detected in post-mortem examination.

These blastomas are really blood spaces, and having Progress and course. no current in them, *thrombosis* often occurs, with the result that the spaces are obliterated. If this process be very general the whole tumour may end in spontaneous cure.

Such spontaneous cure may also result from inflammation of the tumour causing thrombosis.

Partial obliteration sometimes takes place ending in cystic degeneration in the centre indicating an attempt of Nature resulting in partial success of the above process of spontaneous cure.

Hæmangioma Simplex occasionally gives rise to Metastasis may occur. metastases. It has remarkable power of infiltration.

**TREATMENT.**—Treatment of these angiomas Treatment. is very difficult. Two procedures may be adopted, *viz.*:—

(1) **EXCISION.**—Where the growth is amenable to such a procedure this method of treatment should always be adopted as a matter of choice. Very few vessels require to be tied, if the surgeon keeps himself at a safe distance away from the tumour running through normal tissue; in such an operation the bleeding is not much. Even if the actual pathological tissues and cavernous spaces are encroached upon by the line of such an incision, the hæmorrhage can be arrested without much difficulty. The incision should be crescentic at the sides and made by a diathermic knife—a high frequency electric cautery. After the removal of the mass the skin may be drawn at the central line, the edges apposed together, and if the site permits, Halstead's sub-cuticular suture employed.

(2) ELECTROLYSIS.—Where excision is not possible this method is the next best that could be adopted. When the electric current passes through the mass it produces chemical and physical changes in the blood and sets up coagulation in it. Diathermic or High frequency cautery are most useful in this condition.

(iii) Plexiform  
Angeiomata.  
These are  
really  
aneurysms.

### (iii) PLEXIFORM ANGEIOMATA.

Border  
line with  
aneurysm.

As we have stated above, this is more a condition of aneurysm than of angeiomata. The line of demarcation is vague, and therefore the plexiform angeiomata require partial description here. These angeiomata are pulsating tumours of the nature of cirroid aneurysm, or aneurysm by anastomosis. Sometimes the growth is formed by the union of many smaller arteries resembling an arterial nævus.

Plexiform Angeiomata so far as the pathological characteristics are concerned exhibit the dominance of arterial element, although veins and capillaries also constitute partly to form it; the tubular characteristics of which is often lost. The growth is usually met with in young people. The *sites* of incidence are characteristically on the scalp at the occipital and temporal aspects where they usually grow. The aneurysmal varieties are found, (a) in the interior of bones, (b) in some forms of pulsating exophthalmos, and (c) in the scalp; described in greater detail under Aneurysm. *Vide* Volume V.

The tumour is usually soft and compressible. It is pulsating and is characterized by marked bruit.

The growth is usually covered by thinned out skin, and is liable to two serious conditions; *viz.*, (i) infection, and (ii) fatal hæmorrhage.

Clinical  
Features.

CLINICAL FEATURES.—The presence of a vascular pulsating tumour which refills quickly on withdrawing the compression is its characteristic feature. The rate of growth is variable, and the patient complains of a constant headache. The adjacent bones may be affected by pressure atrophy.

**TREATMENT** is very unsatisfactory. If it is Treatment. possible complete excision may be successful in some instances; but in performing such operations the incision must run well away from the area of the tumour.

Electrolysis combined with the control of the external carotids well effected by ligaturing them may yield satisfactory result in grave cases.

## 2. LYMPHANGEIOMATA

2. Lymph-  
angeiomata.

Consist of growths composed of newly formed lymphatics; or of a mass of dilated lymphatics, in the latter condition they are known as *Lymphangiectasis*. In the former instances they may become cavernous or cystic. That is to say, three groups of cases are found in this type of Angeiomata also; *viz.* :—(i) the Capillary Three groups,  
*viz.* Lymphangeiomata; (ii) the Cavernous Lymphangeiomata; (iii) the Lymphangiectasis, or a mass of dilated lymphatics. All these may be congenital or acquired.

### (i) THE CAPILLARY LYMPHANGEIOMATA

(i) The  
capillary  
lymph-  
angeiomata.

Capillary Lymphangeiomata develop in the skin, and are congenital in origin. It is also called Lymphatic-Nævus, as it consists of collections of dilated lymph vessels. It is found as a patch sometimes of large size usually of dull yellowish-brown colour after birth. The patch increases considerably with the growth of the child.

In extent it may sometimes develop to a large size and proportion, and sometimes may be associated with lipomatous growths of the underlying connective tissues, when it is described as **Nævo-Lipoma** described below.

Its **surface** may be smooth or wart-like. On examination with an ordinary lens vesicles could be observed on the projecting points.

The **colour** varies according to the contents which may be lymph or any degree of mixture with blood.

The diagnosis may be difficult owing to the absence of blood.

The **sites** of incidence,—generally these growths develop on the neck or thorax.

The growths are composed of *newly formed* lymphatic capillaries which retain their tubular character, separated by fatty connective tissue. True lymphatic blastomata occur in the subcutaneous tissue and muscles and when these capillary cysts develop under the subcutaneous tissues they may still proliferate and manifest signs of malignant nature. But true lymphatic blastomata are rare. Large cysts of cavernous types, described below are found invading and infiltrating in the surrounding tissues, which are rarely well defined and sometimes burrowing widely. The tumour appears like soft spongy swelling which when cut exudes a large quantity of lymph which sometimes may be mixed with blood.

**Treatment.** **TREATMENT.**—Radical excision is the only treatment that should be attempted. Cauterization may also be successful.

**Nævo-Lipoma.** **NÆVO-LIPOMA.**—In some rare forms of Nævus fatty tissues are blended in abundance. This kind of growth is called Nævo-Lipoma.

Nævo-Lipoma is congenital in origin, or they may appear early after birth.

In appearance these tumours present a lobulated swelling. The consistence is doughy in nature like fatty tumours. The surface exhibits a few dilated veins and capillaries.

By pressing on the tumour its size can to a certain extent be reduced. No thrill or pulsation or bruit could be detected inside, and the tumour, excepting its bluish hue and dense texture, resembles a lipoma closely.

Treatment is excision, which is not difficult to perform.

**(ii) CAVERNOUS LYMPHANGEIOMA.**

(ii) Cavernous  
Lymphangeioma.  
Border  
line with  
cysts.

It is a tumour composed of structure comparable with that of the Cavernous Angeioma. As in the Hæmangeiomata there are some tumours which are on the border line of aneurysm so in this type of Lymphangeioma it is sometimes difficult to differentiate it from a lymphatic cyst.

A lymphatic cyst is neither a cystomata nor a Cavernous Lymphangeioma. A cystomata is formed by the active proliferation of the matrix containing lymph enclosed in cystic spaces; whereas Cavernous Lymphangeioma is a growth composed of newly formed lymphatics, which do not retain their tubular structure. The diffuse enlargement of the lip and tongue known as **Macrocheilia** and **Macroglossia** described below are of this nature of lesion.

These lymphangeiomata may grow at any part of the body, and may co-exist with the capillary variety.

In **size** they vary from a split pea especially when they grow on the skin, to large cyst like swellings when they develop in structures like the tongue or lips, *e.g.*, **Macroglossia** and **Macrocheilia**.

They may be unilocular or multilocular, isolated or scattered, or may develop in groups like herpes from which condition it is sometimes difficult to differentiate in the acquired variety; as in some instances the lesion manifest itself as groups of small vesicles. The only diagnostic point against herpes is the absence of the inflammatory redness round the vesicles in lymphangeioma.

The content of these growths is true lymph. When the cyst is opened a condition of lymphorrhœa, that is flow of lymph fluid lasting for a considerable period, takes place. This is more pronounced on the prepuce and the inner side of the thigh.

**TREATMENT.**—Excision is the best method. In some cases the wound should be left packed with gauze to encourage healing by granulation.

Macro-  
glossia  
and Macro-  
cheilia.  
Border  
line with  
hyper-  
trophy.

### MACROGLOSSIA AND MACROCHEILIA.

Sometimes individuals are met with abnormally large tongue which are called **Macroglossia**; and similarly in some individuals abnormally overgrown lips are found which are called **Macrocheilia**. These are congenital conditions due to obstruction of the lymphatics with associated overgrowths of the connective tissues intervening between them, the underlying process being that of the nature of Cavernous Lymphangioma.

These conditions as stated above are congenital in origin and they exhibit the nature of fibrous hyperplasia with dilatation of the lymphatics. In some cases Macroglossia may develop from fibromatosis of the nerves.

**TREATMENT.**—It is better to leave these conditions alone, as no treatment improves them.

(iii) Lym-  
phangiec-  
tasis.

### (iii) LYMPHANGIECTASIS.

(a) Simple.

(a) **SIMPLE LYMPHANGIECTASIS.**—When the normal lymphatic vessels are dilated keeping their continuity with the normal lymphatic circulation the condition is called Lymphangiectasis. It may be acquired and also congenital; but congenital cases are more frequently met with. The latter condition is perhaps due to some anti-natal inflammatory condition or some abnormality in the development of the lymphatics. It is usually situated in the skin of the face and neck. There must be a mass of dilated lymphatics to constitute a Lymphangiectasis.

(b) Cystic.

(b) **CYSTIC LYMPHANGIECTASIS.**—A simple lymphangiectasis when combined with an active secretion into the lumen of the dilated lymph capillaries develops into a cystic type of Lymphangiectasis. The dilatation of the tubules is the direct sequence of some kind of obstruction to the outflow of the lymph. **Cystic Hygroma** of the neck is the best known example of this condition. A Hygroma is composed of a collection of many large

Cystic  
Hygroma.

spaces containing clear lymph lined by endothelium and bounded by fibrous tissue.

TREATMENT consists of excision, for the details Treatment of which, *vide* Surgery of the neck.

### 3. SEROUS ENDOTHELIOMATA.

3. Serous  
Endothe-  
liomata.

More correctly this type of Endotheliomata may be described as Endotheliomata arising in connection with serous membrane. But the first two varieties, *viz.*, the vascular and lymphatic groups are described in a way after which description of this type as suggested above would help the student to remember this group more easily.

Serous membranes are met with, in the cranial and spinal cavities,—the membranes of the central nervous system; in the thoracic cavity the pericardium and the pleura; in the abdominal cavity the peritoneum, and its scrotal process the tunica vaginalis.

Serous  
membranes.

The most important members of this group are the Endotheliomata arising in connection with the **arachnoid**. Majority of the instances grow from the cranial membrane from its inner side. A few grow from the spinal or vertebral canal, and a fewer still, grow from the sheaths of the cranial nerves.

The  
majority  
arise  
from the  
Arachnoid.

The instances of Endotheliomata arising from the pleura and peritoneum are occasional.

Tumours arising in connection with the endothelial cells are usually recognized by their own peculiar features. A normal endothelial cell is a large flat cell with a large oval nucleus. But in a new growth it may become polygonal or cubical or even elongated; where it may occur in the following forms: (i) as a **lining**, *cf.*, those in the vessels, (ii) as concentric masses, often exhibiting a **whorled** arrangement, (iii) as solid **columns** of cells.

The endo-  
thelial  
cells.

- (i) Lining,  
and
- (ii) Whorl  
arrange-  
ments.
- (iii) Solid  
columns.

So that we have to deal with Endotheliomata arising in connection with serous tissues in the following situations, *viz.* :—

Endothe-  
liomata.

(1) Cerebral.  
membrane.

### (1) CEREBRAL MEMBRANES.

(i) Meningioma.

(i) Meningioma or as they were called Endothelioma.

(ii) Psammoma.

(ii) Psammoma.

(2) Pleural.

### (2) PLEURA AND PERITONEUM.

Not common. These are usually flattened or nodular growths, or sometimes spreading diffusely over the membrane.

(3) Capillary vessels.

(3) Arising from **capillary vessels**, and not from the lining cells of the membrane.

The Endotheliomata of this group that is arising from serous tissues consist of endothelial cells exhibiting various forms and appearances; *viz.*, sometimes the cells are short and plump, sometimes thin and drawn more or less spindle-shaped, having characteristically whorled arrangement. Some of the whorls are limited in their extent, *e.g.*, only closely grouped around capillary vessels; or sometimes more generally extended to form part of a wider system. As we know giant cells in inflammatory reactions are mostly formed of endothelial cells, in tumours also arising from endothelial tissues, multinucleated giant cells are formed. At other places the cells in the middle whorl appear as packed together in solid columns where no central capillary may be found.

Meningioma.

**MENINGIOMATA.**—Till recently these tumours were described as Endotheliomata arising from the dura mater. At the outset it must be remembered that they do not arise from the dura but do so from the **arachnoid** villi and originate from the clusters of **Menigocytes** which cap the villi.

Changes in them.

The older growths exhibit fibrous changes in places. Calcareous deposition is prominent in most of them. The meningioma cells arrange themselves in concentric whorls. These whorls look like grains of sand when calcification occurs in these cell whorls. Sometimes

Calcification.

small areas of true bone formation are met with. These True bone. partly calcified Meningiomas are called **Psammoma**, described below.

In appearance a Meningioma varies. It may be sessile or pedunculated; or if it grow at the base of the skull it may assume the form of a flat plaque. Its surface may be smooth or in some instances it may be lobulated.

Meningiomata usually arise in close relation with the venous sinuses of the cranium most commonly of the anterior and middle fossæ, the commonest sites being the cavernous sinus, the olfactory groove, the sagittal (longitudinal) sinus, the transverse (lateral) sinus and the falx cerebri.

Meningiomas arising in the spinal column are often associated with the origin of the spinal nerves, more especially the posterior root.

Meningiomata are locally malignant causing fatal results by the damage done on the brain by pressure. They may penetrate or push aside the brain slowly deforming it by depression, at the same time may progress for years and attain considerable dimensions without giving rise to any localizing signs or symptoms of marked intracranial pressure. They may encroach upon the dura and penetrate the skull.

The latest accepted view is that 50 per cent. of the cases appear to give a definite history of trauma as the predisposing cause.

Most of the Meningiomas were formally described as Sarcoma.

**TREATMENT** consists of extirpation which can Treatment. be done without much difficulty by shelling it off.

**PSAMMOMA BODIES.**—Sometimes as described Psammoma above the tumour cells spindle-shaped in appearance, bodies. may group together to constitute small nodules arranging themselves in laminated form layer by layer in which the cells have become infiltrated with calcium salts. These

nodules are especially described as **psammoma** bodies. These nodular growths are hard and calcareous throughout, and these calcareous bodies are bound together by a delicate connective tissue net work, the latter is sometimes highly vascular, where the endothelium of the capillaries exhibits a tendency to proliferation.

Some Psammoma bodies are perhaps angiomas in which calcium deposit has occurred as a secondary calcareous infiltration in capillary thrombi, or in the walls of the smaller vessels, or in groups of proliferated endothelial cells.

Psammomata.

**PSAMMOMATA.**—Psammomata are tumours composed chiefly of Psammoma bodies described above. Psammoma is a meningioma with calcareous changes. As described above it is a small nodular and hard growth which is calcareous throughout. On superficial examination, the cut endothelial columns resemble "cell-nests", *vide* carcinoma.

Endothelioma in the pleura.

Endotheliomata in the **pleura** are not common. They occur as nodular and flattened growths often widely spreading over the surface of the membrane. In their histology they appear like epithelial tumours. They are composed of endothelial cells enclosed in a fibrous stroma, exhibiting an irregular acinous arrangement.

Some tumours were incorrectly described under Endotheliomata. They cannot be called pure Endotheliomata.

Some tumours, *e.g.*, the following are described under Endotheliomata without full justification. They cannot be called Endothelioma to its true sense.

(i) Potato tumour.

(i) **POTATO** tumours in the neck in connection with the carotid gland. This form starts from the

**sheath** around the vessels which normally forms their cellular investment and therefore is often described as:

(ii) **PERI-THELIOMA**.—Perithelioma may occur in any tissue, but are most commonly met with in the parotid gland, described as:

(ii) Peri-thelioma.

(iii) **MIXED PAROTID** tumours. Although some endothelial elements are found in these tumours, in the parotid they contain various other cells such as those of fibrous tissue, cartilage, and various other admixtures of typical cells. *Vide* Teratoma. Page 273.

(iii) Mixed Parotid Tumours.

(iv) **GIANT-CELLED** Tumours or **myeloids** of the tendon sheaths exhibit some endotheliomatous nature and are therefore sometimes included under Endotheliomata. It is doubtful whether like other Myeloids they are blastomas at all. *Vide* Blastomatoids. Pages 23, 29, 63.

(iv) Giant-celled tumours of the tendon sheaths.

## II. EPITHELIAL TISSUE TUMOURS

### PAPILLOMATA AND ADENOMATA.

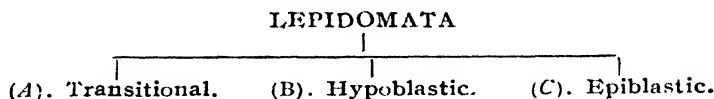
Papillomata and Adenomata belong to **Lepidomata** class of tumours. That is to say they arise from *lining* membrane tissue. The Lepidomata which are non-malignant in nature are described as Papillomata or Adenomata according to the nature of the arrangement of cells and the stroma. Ziegler describes them under one term **epitheliomata**—a term sometimes used for carcinoma by other writers.

II. Epithelial tissue tumours. Papillomata and Adenomata.

We have already seen *vide* Adami's classification in the previous chapter that Lepidomata may be, (a) Primary, and (b) Secondary or Transitional. They are called primary when the cells have descended directly from the original epiblast or hypoblast during development; or secondary when they have passed through a mesoblastic

Primary and Secondary Transitional.

stage in the course of their career. So that we may arrange them in a Table as under:—



A. Papillomata and Adenomata of organs, the constituent cells of which had passed through a mesoblastic or transitional stage are the following:—

Papilloma and Adenoma of Kidney.

"	"	"	"	Testicle.
"	"	"	"	Ovary.
"	"	"	"	Urogenital Duct.
"	"	"	"	Uterus.
"	"	"	"	Serous Membranes <i>e.g.</i> ,

pleura and peritoneum, described as Mesothelioma.

B. Papilloma and Adenoma of organs, the constituent cells of which are of hypoblastic origin, are the following:—

Papilloma and Adenoma of Digestive Tract.

"	"	"	"	Respiratory Tract.
"	"	"	"	Thyreoid.
"	"	"	"	Pancreas.
"	"	"	"	Liver.
"	"	"	"	Bladder.

C. Papilloma and Adenoma of organs, the constituent cells of which are of epiblastic origin are the following:—

Papilloma of Surface Epidermis.

"	"	"	"	and Adenoma of Sweat gland.
"	"	"	"	Sebacous gland.
"	"	"	"	Mammary gland.
"	"	"	"	Salivary gland.

The arrangement of the Papillomata and Adenomata as tabled and listed above suits our purpose in connection with the classifications and Tables set out in our T. T. T. Table, No. XIX, *vide* Chapter II of this Volume.

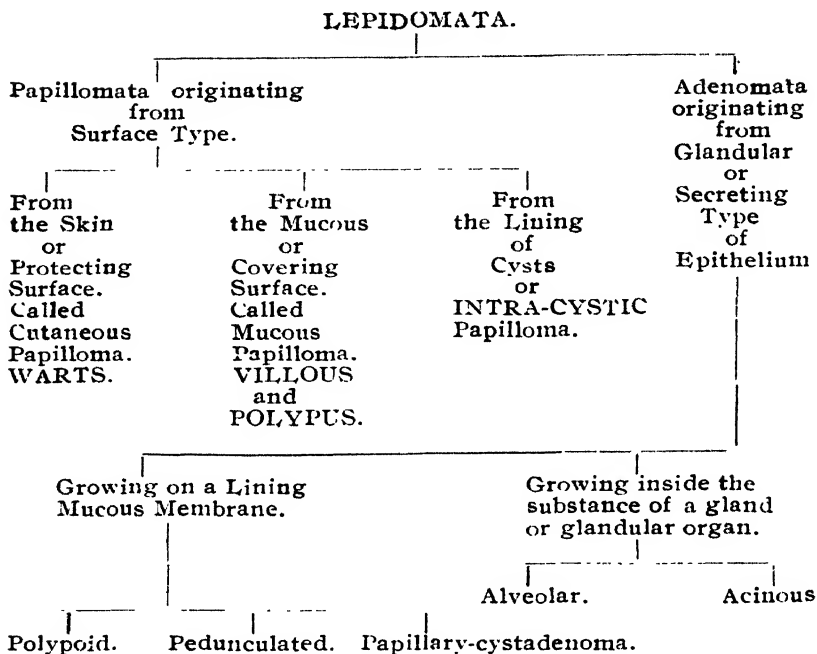
As stated above Papilloma and Adenoma are growths whose processes of proliferation are just the same excepting that the former is concerned with the lining epithelium having no secretion, and the latter develops from glandular type of epithelium secreting out some kind of fluid. That is to say, these kinds of Lepidomata or epithelial tumours may be divided into two main forms, *viz.* :—

1. Those of the Surface Type or Protecting Type of Epithelium.

2. Those of the Secreting or Glandular Type of Epithelium.

Before proceeding to a separate description of the two types of these epithelial tumours, we shall discuss some common points and features, and also some peculiarity of particular histological structures which are concerned in both of them; and it would be convenient to study them at this stage. It is according to their *mode of growth* and the nature of the epithelium these two main types of epithelial tumours are described. That is to say; (i) those which correspond to the surface or covering or lining cells are called Papilloma; (ii) and those which correspond to the secretory or glandular organs are called Adenoma. Otherwise Papillomata and Adenomata agree in their general properties although they exhibit differences in the mode of growth and behaviour depending upon their individual structure and kind of cells of which they are composed. Each of these two types again are divided into groups according to their origin, *viz.* :—

What is a  
Papilloma?  
What is an  
Adenoma?



The main points, to avoid confusion which may arise in the mind of the beginner, are to remember that, (a) all epithelial cells do not secrete, (b) all epithelial cells are not of simple type, and (c) all epithelial cells which constitute a surface for protection or covering are compound. For instance, the wall of the bladder is lined by mucous surface but the epithelium are of transitional surface type (not squamous), which gives rise to villous type of Papilloma; whereas in the uterus the mucous surface will give rise to Pedunculated type of Adenoma. On the other hand the mucous membrane of the alimentary canal may give rise to Colitis Polyposa, in other words mucous type of Papilloma, as well as Polypoidal or Pedunculated Adenoma. The main difference between the two, being the following:—

(i) Papilloma does not concern itself with secretion; whereas Adenoma secretes a fluid, or at least tries to do so.

(ii) In the Papilloma the cells are situated externally adopting a uniform arrangement around an internal core of connective tissue; whereas in the Adenoma they form either *tubules*, or *solid masses* which are embedded in, and surrounded by, the connective tissue stroma.

(iii) But then in the latter group where they form into tubules it is not uncommon for them to dilate into cysts, where the lining epithelium may project into the cavity taking a connective tissue core inside them for blood supply, in the same way as an ordinary Villous Papilloma would grow on a mucous surface. In such circumstances we describe them as **papillary cyst-adenoma**. Vide Table in the previous page.

(iv) These two types of epithelial tumours are non-malignant, they progress slowly, they never infiltrate, although they compress the surrounding neighbouring tissues and organs. Metastases are unknown in them.

(v) The cells closely conform the normal types, and although sometimes they proliferate to an exuberant growth the process is never so uncontrolled as to cause difficulty in determining the nature of the tissue from which the growth originated.

(vi) The blood vessels which develop in the stroma are always well-formed and their walls are always separated from the tumour cells by a definite layer of connective tissue however delicate.

(vii) Degenerative changes are rare in epithelial histioma. But the cells may be excited to copy a secretion which may be mucoid or colloid.

(viii) Malignant transformation of Papilloma is common as it may assume malignant character at any time; and owing to its being exposed to injury by pressure or friction, hæmorrhage may occur and ulceration may result from any mechanical irritation.

(ix) Inflammatory change inside these tumours following infection with septic organism is not an unfrequent occurrence; and these tumours become more liable to be so for their situations, being either on the tract of a septic passage or inside an organ which may easily be infected.

(x) Fatty degeneration of the cells and myxomatous and hyaline changes in the stroma may occur. Calcareous changes may also take place in an adenoma or papilloma.

We shall now take to describe these two types of tumours individually.

## 1. PAPILLOMA.

Papilloma. In its true sense the term Papilloma was used to describe the warty growths on the skin. It was so called because it is a neoplasm which reproduces the structure of the *papillæ*. It is most commonly met with on the skin, as a **wart**. On muco-cutaneous junctions, *e.g.*, at the anus or labiæ and also on mucous membranes, lining the mouth, pharynx, larynx, vagina, anus, prepuce, that is to say, all the areas where actual papillæ are met with Papillomata are described. The term papilloma is of course now used for tumours or any villous excrescence of an epithelial surface where actual papillæ may not exist but wherever the growth looks more or less cauliflower shaped and resembles in structure to the normal papillæ.

All varieties of papillomata resemble a wart in structure although they grow in various forms.

The structure of all varieties of papilloma more or less resembles an ordinary **wart** with the exception of their form; *e.g.*, some of the individual growths look like threads, some like stumps or finger-like processes, others resemble mushroom-like growths, and there are others again which are distinctly polypus-like in appearance. Some look like a piece of cauliflower. In the bladder these villous growths bear a striking resemblance to certain seaweeds.

WART.—An ordinary wart so often met with independently or in association with other growths such as elephantiasis of the legs consist of the following **structures**, viz. :—

The structure of a wart

(i) A central connective tissue core of mesoblastic origin. This serves as a stalk, which may be branched; the branching being repeated many times resembling the arborescent arrangement of a plant. In a papilloma this stalk is more cellular than such a core of the normal papillæ, sometimes it may be myxomatous especially in a slow growing tumour. This stalk contains blood vessels. It is important to remember this vascular construction, as the more elongated or hair-like or villous the papillomata become, the more tortuous tuft-like mass of delicate blood vessels of innumerable number are exposed, which readily give way leading to uncontrollable hæmorrhage, *e.g.*, in the bladder. In Papillomata these blood vessels inside the core are more dilated than normal, but they always possess a definite wall.

(ii) Covering the stalk is a layer of epiblastic tissue consisting of many layers of superimposed epithelium which is continuous with the surface epidermis or the mucous covering of the part of the body where the growth originates; but usually such layers are thicker in the deeper portion. In an ordinary wart three distinct layers can be distinguished, viz.; (a) a broad deep layer of 'prickle cells' underneath, whose individual cells are rounded in appearance, where **eleidin** granules sometimes begin to appear, (b) an intermediate layer of flattened cells in process of transformation into: (c) the superficial or horny layer, or keratinized layer. When the tumour grows the surface becomes irregular and the uppermost surface with the keratin material may dip down and become infolded; some portion of which may separate thus get lost, and be lodged in deeper tissues appearing under the microscope as rounded "cell-nests." *Vide Squamous Carcinoma. Vide p. 209.*

All the above structures can be clearly demonstrated by staining a section of the diseased tissue by picrofuchsin. Stained as such the tissue exhibits the following structure as under:—

Red blood corpuscles stain bright yellow.

Deep layer of epithelium stains brownish.

Fibrous tissue stains pink.

Horny layer stains bright yellow.

Patho-  
logical  
variety.

THE PATHOLOGICAL VARIETIES.—We have already stated that Papillomata may be divided into three groups, *viz.*:—

1. Cutaneous type.

(1) Cutaneous type, or typical wart.

2. Mucous type.

(2) Mucous surface type or typical villous or Polyposa.

3. Intra-cystic type

(3) Intra-cystic type.

Histological varieties of cells found.

Histologically, the following kinds of epithelial cells are found in the various pathological varieties described above, *viz.*:—

1. Squamous epithelium.

(1) Squamous type of epithelium with keratin granules, is found in the Cutaneous type of Papillomata called sometimes Warts or Hard Warts. These are of course compound epithelium, exhibiting the other types of epithelium in the deeper layers.

The epithelium found in the villous excrescence of the bladder are also compound but they are transitional in type, having no keratin layer.

2. Cuboidal epithelium.

(2) Cuboidal or Spheroidal type of epithelium is found in the Papillomata of breast, kidney, prostate, thyroid and other glandular structures where cuboidal epithelium of simple type prevails.

3. Columnar epithelium.

(3) Columnar epithelium is found in the Papilloma of the intestine called Colitis Polyposa, and in the cystic cavities in other tumours as for instance the ovarian cysts.

## CLINICAL TYPES OF PAPILLOMATA.

(i) Papilloma on the skin is called Hard Warts, or Squamous Papilloma. Clinical types of Papillomata.

(ii) Papilloma on Mucous membrane of soft consistence, is called Mucous Papilloma or Soft Warts, including under this condition:—

(1) Villous Papilloma on the bladder wall.

(2) Colitis Polyposa seen in the intestine.

(3) Polypoid Papilloma on the laryngeal mucosa.

(3) Intercystic Papilloma or the Papilloma met with in the ovarian cysts, etc.

Each of the above varieties demands description in a greater detail which follows.

## 1. CUTANEOUS PAPILLOMA.

1. Cutaneous Papilloma.

(i) **HARD WARTS.**—When they occur on the skin they form a hard wart attached to the skin by a broad base especially on parts subjected to friction and irritation. They are covered by squamous epithelium and consist of bundles of hypertrophied papillæ. They sometime extensively proliferate and branch in arborescent manner, forming secondary, tertiary, and innumerable branching papillæ. In common warts the epithelium is keratinized, and sometimes the growth becomes very hard and develops into a horn-like process. The surface of an ordinary wart is finely fissured, but the individual processes are never very long, and are not distinct. A moderate degree of pigmentation is always present, and the tumours are often multiple.

(i) Hard Warts.

(ii) **SOFT WARTS.**—When such squamous papilloma occurs on moist surfaces such as on the prepuce the growth remains soft as the formation of horny layer is imperfect. Laryngeal cord is an exception. (ii) Soft warts.

## 2. MUCOUS OR MOIST PAPILLOMA.

2. Mucous or moist Papilloma.

(i) **VILLOUS** Papilloma develops on the mucosa of the urinary bladder and the pelvis of the kidney. They (i) Villous Papilloma.

are composed of a connective tissue vascular core, covered by many layers of transitional epithelium forming tufts of slender vessels which readily bleed causing dangerous hæmorrhage. These very often become malignant, or grow in association with malignant growths.

(ii) Soft  
Papilloma.

(ii) **SOFT** Papilloma is seen in the intestines. It consists of a solitary thick finger-like process. There may be hundreds of them growing at different sites, when they are described as **Colitis Polyposa**. In the larynx and on the tongue soft papillomata develop which are intermediate in structure between the cutaneous and mucous types.

3. Intra-  
cystic  
Papilloma.

### 3. INTRA-CYSTIC PAPILLOMA.

Sometimes Papillomata are met with inside other tumours such as the ovarian cysts, duct carcinoma of the breast, other dilated ducts, etc. These are covered by columnar epithelium.

Blastoma-  
toids of  
Epithe-  
lium.

### BLASTOMATOIDS OF EPITHELIUM.

Callosity.

1. A simple localized collection of epidermal cells is called a *callosity*. Sometimes such callosities may develop to a considerable size.

Keratoma.

2. A localized accumulation of the horny layers of the epidermis is called a Keratoma or a Horn. These Keratomas grow to a considerable size. They often develop on the forehead or scalp. They sometimes arise in connection with sebaceous tumour.

Paraffin or  
Bilharzia  
Irritations.

3. Sometimes papillomatous growth develops in connection with chronic irritation resulting in an excessive overgrowth of the papillæ, and in some instances they are infective inflammation in nature. These sometimes undergo a malignant metamorphosis and become carcinomatous. They are often met with on the hands, and fingers.

4. Condylomata is a typical papillomatous blastomatomatoid process for the description of which the reader is referred to Syphilitic Ulcers. They are also described as venereal warts. Condylomata and Coccidiosis.

**TREATMENT** is complete extirpation. The procedure and the steps of the operation varies according to the sites from which the growth develops. For the descriptions of which *vide* surgery of the regions concerned. Treatment.

It must be understood that considering the clinical cases of blastomatomatoid growths and blastomata with regard to the skin which are commonly met with, an authentic blastoma of this type is a rarity and most of the clinical cases of papillomatous growths met with are blastomatomatoid processes. For a detailed description *vide* Chapter I, on Blastomatoids. P. 45.

## 2. ADENOMA.

Adenoma is a neoplasm composed of incompletely formed glandular tissues. Adenomata tend to reproduce the structure of secreting glands or gland-like organs from which they grow. We say gland-like because although they resemble glands in structure they are incapable of producing the characteristic secretions and they cannot reproduce the ducts to discharge them. Moreover, the alveoli are less perfectly developed or sometimes may be entirely occupied by only several layers of epithelial cells forming a spherical solid mass encapsulated all round. Thus the formation itself is atypical that is verging towards malignancy. An Adenoma may have an acinous arrangement, that is a tubular structure lined with columnar epithelium when it grows on a mucous lining, or it may have an alveolar structure lined with spheroidal epithelium, when it grows in the substance of a solid gland; *e.g.*, liver, thyreoid, breast, etc. Adenoma.

According to its site of growth is called Pedunculated or Intraglandular.

That is to say, according to either it grows on a mucous lining such as the alimentary canal, or inside a solid gland such as the breast, its form is changed. In the first condition it grows freely at all sides keeping a pedicle for supply and therefore its shape becomes somewhat irregular and polypoid and it is therefore always **pedunculated**. In the second condition when it grows inside a gland it is suppressed at all sides equally, and therefore grows like a mass more or less **spherical** in form. It is then described as **intraglandular**.

Therefore, according to the parent organ or gland or tissue the structure of the Adenoma largely depends. For instance when it grows inside, in the substance of a gland such as the liver or the sebaceous gland of the skin, the cells constituting the tumour exhibit no attempt at the formation of acini or a tube; but simply grows together in a mass in a chamber in the form of solid alveolus or trabeculae and adopt an **alveolar** formation. But when an adenoma arises from a racemose or tubular gland a tubular **acinous** arrangement is the rule. It may not be possible to find the distinction very strictly in all cases, as one kind may exist in the other.

Alveolar.

Acinous.

In a simple tubular gland as for instance in the intestine, the secreting epithelium is *columnar* in type. In compound or racemose glands as are the cases in the thyroid and breast the secreting epithelium is *cuboidal* or *spheroidal*; and it is according to this histological peculiarity an Adenoma is described as Columnar or Cuboidal Adenoma. Pedunculated Adenomata are generally *acinous* and composed of columnar cells and are therefore called Columnar-celled Adenomata; *e.g.*, of the intestinal tract, uterus, etc.; and Intra-glandular Adenomata are generally *alveolar* or spherical, and are composed of spheroidal or cuboidal cells, and are therefore called Cuboidal or Spheroidal-celled Adenomata, *e.g.*, the thyroid, breast, prostate, etc. But the contrary of this rule may not be the case, for acini may be found in the tumours of the solid alveolar group.

The newly formed cells, in the majority of the instances of Adenomata are capable of carrying on their inherent normal functions of secreting out a certain quantity of their fluids to a considerable extent, *e.g.*, we find *colloid* substance in the Adenoma of the thyreoid, *mucin* secreted out from the goblet cells of the intestinal growths, *bile* produced in the Adenomata of the liver, etc.

In some instances the secretion may be so abundant as to proportionately modify its consistence, for the arrangement and construction of the acini being always defective and irregular, the removal of the secreted fluid is not provided for; and although the growth is surrounded by the mother gland the latter does not show any tendency to allow its own normal duct to be used for the purpose; with the result that the newly formed or neoplastic acini become distended with the retained secretion, the tumour being ultimately converted into a collection of **cysts** especially described as **Cyst-Adenoma**.

May form  
Cyst-  
Adenoma.

But we have found in a certain variety of Papilloma that the epithelium proliferates abundantly inside a cyst described as **Papillary Cyst-Adenoma**. Here the connective tissue support is thrown into folds projecting into the lumen which resembles the papillary growth known as Papillary-adenoma. The cells lining the cyst gradually atrophy from hydrostatic pressure of the secretion, or they may continue to proliferate into the lumen giving rise to what is known as Papillary Cyst-Adenoma.

Papillary  
cyst-  
adenoma.

Adenoma may be single or multiple, usually encapsuled and connected with the parent glands by a pedicle, through which the vessels enter, or through its connective tissue capsules in an intra-glandular variety.

May be  
Single or  
multiple.

Adenoma is composed of glandular epithelial structure supported by connective tissue stroma, containing the blood vessels, the whole mass being enclosed in a fibrous capsule as described above. The glandular structure is atypical. It consists of spheroidal or cuboidal epithelium may be columnar according to the parent

General  
pathologi-  
cal struc-  
ture.

gland. The cells do not secrete, and the acini are irregular in size shape and arrangement. Sometimes cystic spaces are formed which in some instances turn the tumour into cyst-adenoma as described above. The connective tissue stroma grows more abundantly than in the normal glands and sometimes may be so abundant as to form Fibro-adenoma, or better called Adeno-fibroma to be described. *Vide* below.

The tubules or acini of an adenoma develop in the same way as the tubules are primarily evolved, or as they grow during pregnancy in the mammary gland, namely, by the budding out of new ducts from previously existing ducts. In some instances a solid rod of cells may all be observed to be in the process of cell division resulting in the formation of a lumen, which in its mode of development resembles the growth of new capillaries in granulation tissue.

The process of cyst formation is described under the adenoma of the ovary in a greater detail where such cystic changes are mostly observed.

Adenomata usually grow in connection with existing glands, or pre-existing glandular organs arising from misplaced rudiments, or very rarely, independently of these.

Fibro-  
Adenoma  
or better  
Adeno-  
fibroma.

**ADENO-FIBROMA.**—We have already described how epithelium always requires some connective tissue to form tumours. In some instances this association of compound structure constituted mainly of an admixture of fibrous tissue may grow to such characteristic prominence, *e.g.*, in the breast where they are mostly seen, that instead of describing them as Adenomata they should be described as Adeno-fibromata. In Adeno-fibromata associated with a hyperplasia of glandular element there is a progressive growth of fibrous-tissue. Usually either the glandular epithelium or the fibrous tissue is the seat of the original progressive proliferation, and in the majority of the clinical instances it is the

fibrous tissue which starts or dominates. It is in very rare instances these tumours are truly compound growths. Modern view is that these tumours should be described as Adeno-fibromata instead of Fibro-adenomata.

Two types of Adeno-fibromata are seen, *viz.* :—

Two types.

(i) Peri-canalicular Adeno-fibroma.

(i) Hard or Pericanalicular.

(ii) Intra-canalicular Adeno-fibroma.

(ii) Soft or Intra-canalicular, or Cabbage like Cyst-adenoma. Cabbage type is seen in the breast. Cauliflower type or papillary cyst-adenoma occurs in the ovary.

In the first type, the fibrous tissue markedly grows around the gland acini. In the second the fibrous tissue projects into the spaces, the tubules become stretched and drawn out by the general over-growth, showing a marked tendency to the formation of cysts in which the proliferating spheroidal cells with ingrowths of connective tissue project in long folds, presenting on section an appearance very similar to that seen on the surface of a cut cabbage, forming a cyst-adenoma. It is quite different from the cauliflower like appearance of a papillary cyst-adenoma. This cauliflower type of cyst-adenoma occurs mostly in the ovary. The cabbage type occurs in the breast.

**BASEMENT MEMBRANE.**—It must be remembered from now that it is the basement membrane which is the most important structure determining the life and death of the patient. It is composed of condensed connective tissue upon which rest the epithelial cells arranged in a single layer. Multiplicity of layers lining the acini may occur which may not be a sign of malignancy, but absence of basement membrane is a sure sign of it.

The basement membrane is the most important structure.

**CLINICAL TYPES.**—According to the histological character of the secreting epithelium as Adenoma is composed of it is described as:—

Various clinical types. According to the histological character of the cells it is called.

### 1. Columnar celled Adenoma.

## 1. COLUMNAR-CELLED ADENOMA.

In a simple tubular gland the secreting epithelium is columnar in type, and they are met with in the glands of the skin, intestinal tract, uterus, ovary, and in infants in the stump of the umbilical cord. And therefore at these sites we come across with the following varieties of Adenomata, *viz.* :—

### (i) In the Skin.—

### Sebaceous Adenoma.

**SEBACEOUS ADENOMATA.**—Are mostly seen on the scalp or labium majus. True neoplasms of this type are rare. They appear as small nodular tumours attached to the skin and red in colour; exhibiting small cystic spaces on section. Microscopically, they consist of glandular structure of racemose type lined with columnar epithelium. The cells are large, the protoplasm has a waxy appearance and therefore stain feebly.

Similar tumours may occur from the **sweat glands** and **hair follicles** also, although they are very rare. Adenomata in connection with sweat glands are found on the skin of the body, but Adenomata of sebaceous glands are met with on the scalp as described above. Retention cysts of the sebaceous glands of the scalp are called **wen**. Many of these so-called wens are of the nature of cyst-adenoma. It may be remembered that in a normal sebaceous gland the sebaceous material is formed of a kind of degeneration of fatty nature of the lining cells.

### (ii) In the Intestines.—

### The Intestinal Tract Adenoma.

## THE INTESTINAL TRACT ADENOMATA.—

These arise in connection with the glands of the gastric or intestinal mucosa. They may grow from the mucosa of the stomach, small intestines, large intestines and rectum, in the form of red easily bleeding polypoid masses.

Various forms are met with, *viz.*, the "**gland polypus of the rectum**," the **polypoid form**, and the **tubular form**. In the latter condition it may protrude on





GLAND POLYPUS OF RECTUM.

the surface as a soft test-tube-like mass composed of the ordinary tubular glands found in the intestines, or the Leiberkuhn's follicles.

Adenomata in the intestines are very liable to become malignant and when they become so they closely resemble columnar epithelioma. In a non-malignant condition, the proliferation of the epithelial cells **does not extend beyond the basement membrane** into the connective tissue, or pass on to invade the muscular coats of the bowels; and by the absence of this infiltration they are distinguished from carcinoma.

It is usually found in the stomach or any part of the large intestines and rectum. They are often attached to the mucous membrane, become pedunculated sometimes attached only by a thin stalk containing the vascular supply forming one or more polyps. *Vide* photo plate No. XL.

Bleeding from the rectum resembling piles, intussusception of the intestines are often caused by these adenomata.

Below the duodenum adenoma arises in connection with Lieberkuhn's follicles, and presents the appearance of a finger-like process which may undergo colloid degeneration.

(iii) In the Ovary.—

### ADENOMA OF THE OVARY.

**Adenoma of the Ovary** exhibits some peculiar features; they are **compound** and **cystic**. They are generally described as **Compound Cystic Ovarian Tumour**, or **Papillary Cyst-Adenoma**.

Adenoma of the Ovary. These are really cystomata, and not cysts.

The tumour consists of newly formed glands spaces lined with epithelium which form into cysts being filled with a serous or semigelatinous material. Ovarian tumours grow to a very large size. They are more or less rounded and globular; or some are distinctly lobulated and multiple; the latter is due to their being com-

Papillary Cyst-adenoma of the Ovary.

How are  
these  
cystomata  
formed?

posed of several cysts. These growths at first consist of solid mass of columnar-celled tubules. Wherever these epithelial cells are provided with extra nourishment they proliferate abundantly and secrete into the lumen of the original duct. This secretion distends the newly-formed duct into a cavity. When the rate of proliferation of the epithelium predominates and exceeds that of the distension, the epithelium is thrown into folds resulting in papillomatous or papilliferous projections inside the chamber of the cysts. But if the secretion is more active than the proliferation distension becomes a more dominating factor, resulting in equally distributed hydrostatic tension which meet a uniform resistance at all parts of the wall of the cyst. This exerts upon the cyst to enlarge uniformly at all radii. Any weak spot anywhere in the wall of such a cyst will bulge out, distend and enlarge into a protrusion, which will form a connected chamber as a daughter cyst. This daughter cyst may ultimately separate. Secondary Cystomata may also develop inside a previously formed cyst by the fusion of the tips of adjacent papillary in-growths forming into endogenous cysts. Ovarian cystomata may become carcinomatous or sarcomatous.

All the cysts that are formed do not develop equally, but a few or one of these as a rule may predominate over the others and contain several gallons of fluid. Quite a good number of small cysts may yet be situated at the "pedicle" of the big cyst. The surface of the tumour is smooth. The contents of the cyst are composed of brownish, serous or homogeneous, sticky fluid of semi-gelatinous nature, in which there may be mixed up the debris of degenerated cells and other granules. Under microscope different areas exhibit different stages of cyst formation, showing all grades between the simple mucoid cyst and the complete and well-formed cyst-adenoma, in the different loculi.

The cysts are lined by epithelial cells of columnar or sometimes of cubical variety, consisting of only one

**single layer**; the free exposed borders resembling the characteristic goblet cells of a mucous membrane. The nuclei are clear deeply placed with faintly coloured protoplasm. These cells rest on a basement membrane made of delicate connective tissue. Outside the basement layer is the stroma which is formed of compact fibrous tissue. The intra-abdominal papillary growths and papillary cyst adenomata often tend to become carcinomatous.

Cystomata of the Ovary are seen in the following varieties according to the nature of their contents, Varieties of Cystomata.  
*viz.* :—

(a) The serous cystadenoma, which is usually multilocular and non-malignant. It grows to large dimensions, its walls are lined with **cubical epithelium** and papillary ingrowths are rare. The fluid is serous mixed with a little fat, epithelial cells, and cholesterin crystals.

(b) Pseudo-mucinous cyst-adenoma is generally a unilateral, multilocular, benign, and pedunculated cystoma. This is the most frequent form of ovarian cyst. This form of cystomata is lined with a single layer of **columnar epithelium**. The fluid is mucinoid or **pseudomucinous**. It may grow to enormous dimensions. Due to the repeated proliferation of the stroma many secondary and tertiary cysts and intracystic folds are formed; for the development of this well-formed fibrous stroma the tumour is supplied with plentiful blood.

Cystomata may meet with various degenerations namely, necrotic, hæmorrhagic, pyococcal and other necrobiotic changes.

Although benign they lead to temporary metastatic growths inside the peritoneum if they gain access into it by intra-peritoneal rupture of the tumour. But they soon die out.

(iv) **UTERINE MUCOSA ADENOMATA.**—Uterine Adenomata grow from the endometrium as red pedunculated tumours projecting into the cavity, or

sometimes protruding out through the cervical canal forming a variety of uterine polypus. The condition is rather frequently seen.

Many of these mucous polyps are blastomatoid processes. Some are difficult to be dignosed from adenocarcinoma as both the non-malignant adenoma and the malignant adeno-carcinoma consist of many tubular glands lined with columnar epithelium. It is the absence of infiltration into the muscle coat which establishes its diagnosis. Another variety called **adeno-myoma** arises in connection with the deeper portions of the glands, but tends to spread into the wall of the uterus; some of which showing distinctly local malignant signs exhibiting very poor development of capsule around them.

**Umbilical Adenomata.** (v) **UMBILICAL ADENOMATA.**—These tumours are sometimes seen in the umbilical cicatrix of infants resembling a raspberry, consisting of masses of tubular glands in a mass of mesodermal tissue. They should always be suspected whenever a persistent sanious or serous discharge oozes out of the navel, when on evert-ing the lips of the umbilicus they may be easily distinguished. The pedicle should be ligatured and the mass removed. Sometimes, although very rarely, Adenoma arises from the post-anal gut in the anococcygeal region.

**Spheroidal Celled Adenoma.** 2. **SPHEROIDAL CELLED ADENOMATA** are met with in connection with the breast, thyreoid, prostate, adrenal, kidney, pituitary, or any other gland, where the secreting epithelium of cuboidal type form racemose glands. These are the following:—

(i) **Adenomata of the Breast.** (i) **ADENOMATA OF THE BREAST.**—Adenomata of the breast is a disease of young age; usually occurring between the ages of eighteen and twenty-five years. They are never seen to occur in the male breast. They may be single or multiple, grow slowly, and if not removed may reach an enormous size.

They are clinically exhibited as rounded well-defined nodular mass, freely movable, easily removable encapsulated tumours, arising in the outlying lobules of the breast with which they do not exhibit any connection. They usually become cystic but occasionally may remain solid.

As described above two types of Adenomata of the breast are met with, *viz.*:—

(a) The Pericanalicular Adeno-fibroma.

(b) The Intra-canalicular Adeno-fibroma.

They may become hard or soft and vascular according to the varying degree of proliferation of the cells of the fibrous stroma or glandular epithelium; or may be cystic, or cysts may be almost entirely absent, when they are described as (1) **Pure Adenoma** (2) **Fibro-Adenoma**, or (3) **Fibro-Cystic Adenoma**, or (4) if the cystic cavity is of a large size they are described as **Adenocoele**.

Microscopically, at first a breast tumour exhibits to consist of fibrous stroma composed of connective tissue corpuscles provided with blood vessels. In the substance of the stroma are seen newly-formed gland tubes in various stages of evolution from imperfectly finished tubes to perfectly formed large acini. At first these tubes are branching and irregular and are solid and closely packed with epithelial cells, but at a later stage a central channel is formed and the epithelial cells tend to assume the task of lining the acini. In some areas these channels are distended into spaces or cavities lined by one or two layers of cubical epithelium. Later, this stroma may grow abundantly and form the **Pericanalicular Adeno-Fibroma**. In other instances small masses of fibrous tissue form into rounded nodules under the epithelial layer and project into the acini and thus block it by cabbage leaf-like folds in it, forming the **Intracanalicular Adeno-Fibroma**.

Microscopical appearance.

On the other hand as has already been explained the gland acini may become enormously dilated to form

cystic cavities of varying sizes described as Adenocoele. In the interior of these cysts sometimes papillomatous projections may develop by the proliferation of the connective tissue stroma lined by a single layer of epithelium, which are described as **Papilliferous Cystic Adenoma**. It often becomes doubtful whether such proliferation is of non-malignant nature. In the mammary gland and ovary as described above, such transformation often takes place, and these cases should therefore be handled and treated as malignant growths.

(ii) Adenoma of the Thyroid.

### (ii) ADENOMA OF THE THYROID.

This may be :—

(a) Foetal gland type.

(b) Adult gland type.

Some interesting general features may be conveniently described here regarding the development of glands from the foetal type to adult type, as that will clear many points of confusion in dealing with neoplasms and blastomatoids of some glandular structures such as the thyroid, the breast, and the prostate which resemble each other in many respects in pathology.

If a foetal thyroid is examined with the naked eye it will be observed to consist of remarkable degree of lobulation; under low magnification these lobules are seen to be widely separated by an abundant connective tissue stroma. A foetal thyroid wholly differs from an adult type in structure. At first it consists of a mass of embryonic cells which cannot be differentiated. Later all these cells arrange in tightly packed masses in different alveoli, separated by connective tissue stroma described above. Finally during the last stage of development the cells are grouped in the form of lining or wall of acini. In adult life sometimes solid masses of cells may be found in the interacinous parenchymatous tissue the so-called "foetal rest" which gives rise to Adenoma composed of foetal type of cells

With the advent of puberty and advancing age the acini develop more and more, but the inter-acinar tissue reduces, till little of the latter structure remains, and this inter-acinar tissue rather varies more with different age periods than with different states of glandular activity.

Now when the glandular activity sets in an interesting phenomenon takes place in the structure of the epithelial cells; *viz.*, the epithelial cells **elongate** into high cubical or low columnar epithelium. At this stage the nucleus no longer fills the body of the cell. The blood vessels of the stroma become full and distended showing a tide state.

When the gland is active. The changes.

When the gland is in resting condition the alveoli are distended with dense **colloid** material, the lining epithelium are turned more flattened or of low cubical type, and no congestion remains in the stroma. This resting stage is the storing stage of colloid, in which **iodine** or more correctly, an organic compound containing 65 per cent. of iodine which is especially described as **thyroxin** is stored in the gland which acts as a stimulant in body metabolism. This active principle of the thyreoid if administered into the system exhibits a remarkable power of invigorating the activity of the organism, or supplementing the effects of thyreoid insufficiency as occurs in diseased condition such as myxoedema. It may be remembered that whereas the female breast has got abundant **lymphatic** supply, and it produces milk, thyreoid has an abundant **blood** supply which supplies an internal secretion in the form of thyroxin.

In the resting condition of the gland. The changes.

So that physiologically secreting glands such as the breast and thyreoid show the following definite stages after development, *viz.*, activity,  $\rightsquigarrow$  hyperplasia  $\rightsquigarrow$  hypertrophy + hyperplasia or in other words **adenomatous condition**  $\rightsquigarrow$  finally ending in repose or rest, that is the

stage of involution. For details *vide* Vol. I. Part I. Chap. I.

Any condition of enlargement of the thyreoid is clinically described as **goitre**. It is a strange peculiar fact that the big fours, *viz.*, the inflammations, tuberculosis, syphilis and malignant disease very rarely attack the thyreoid.

So far as the enlargement of the thyreoid or in other words formation and development of goitre is concerned, any physiological stimulus such as puberty, menstruation, pregnancy; or pathological irritation such as bacterial infection; or even psychic disturbance such as shock, may demand greater iodine supply, which irritates it to hyperfunction followed by hyperplasia and hypertrophy.

The adenomatous goitre or in other words **Adenomata of Thyreoid** may arise in connection with the lateral lobes, isthmus, or thyro-glossal duct of the gland. They are met with, as stated above, in two forms, *viz.* :—

Fœtal type.

Adult type.

(a) **FŒTAL GLAND TYPE ADENOMA.**—In this form for some unknown reason the fœtal cells are awakened into activity. It is a single tumour consisting of solid mass of spheroidal epithelium resembling the structure found in developing thyreoid as explained above. It does not develop into any great size. Degenerations are most common in this form of goitre. Hæmorrhage, hyline degeneration, colloid degeneration in hæmorrhagic areas, fatty degeneration, necrosis, calcareous degeneration, and even actual calcification often occur in this form of goitre. This form like the others may be **Toxic** or **Non-Toxic**.

(b) **ADULT GLAND TYPE ADENOMATA.**—These tumours consist of many spaces lined with one layer of flattened spheroidal epithelium filled with colloid





ADENOMA OF THYROID.  
(Showing Toxic Symptoms).

as in a normal gland. Adult type Adenomata are more frequently met with than the former; they are multiple and unlike the previous form they may attain a very large size. One or two cystic spaces may attain a very large size where hæmorrhage may also occur and cause destruction of the epithelium with the septa. Not only the septa with the epithelium may be destroyed by hæmorrhagic degeneration, but in this form the accumulation of colloid may be so great as to constitute the tumour of circumscribed masses of colloid material, each nodule being surrounded by more or less well developed fibrous capsule. The colloid material met with is similar to that of the normal thyreoid gland. The epithelium is not flattened but cubical, or may even approach the columnar type. The tumour being definitely encapsuled, may be more or less easily enucleated. This form of goitre may also be **Toxic** or **Non-Toxic**. *Vide* photo plate No. XLI.

There is no doubt that in some cases of old standing Adenomata malignant changes may take place.

(iii) **ADENOMA OF THE PROSTATE.**—This is a very common tumour which occurs in the male after the age of fifty-five. The neoplasm is of the fibro-adenomatous type, and may be hard or soft as occurs in the breast of the female; the microscopical appearance of the two conditions being so similar that it may be impossible to say under the microscope whether the cut tissue was from the adenoma of the breast or prostate. (iii) Adenoma of the Prostate.

Prostatic Adenomata are definitely encapsuled, the normal portion of the gland being compressed, and flattened around them.

It is yet an undecided question whether prostatic adenoma is not a blastomatoid process.

The most important Adenomata are dealt with here. For their detailed description and Adenomata arising in

connection with other organs the reader is referred to Regional Surgery.

Diagnosis  
of Adeno-  
mata.

DIAGNOSIS.—For differential diagnosis from other tumours and swellings, *vide* Chapter VI of this volume.

Secondary  
and subse-  
quent  
changes.

SECONDARY CHANGES often occur in Adenomata, most of which have already been described. The common secondary changes are: (1) Fatty degeneration of the epithelium; fatty degeneration in the contents of a cyst; cholesterin formation from free fat. (2) Hæmorrhages in the cysts, caused by badly formed blood vessels. (3) Occasionally inflammatory processes may set in. (4) Malignant transformation of the epithelium into carcinoma; occasionally the connective tissues undergo a sarcomatous change.

The malignant Adenomata have no basement membrane as in simple Adenomata. They infiltrate into the neighbouring tissues, implicate the lymphatic glands, and give rise to secondary growths. They exhibit other signs and symptoms of characteristic malignant features, *e.g.*, cachexia, etc. Moreover the epithelial cells in non-malignant adenoma are uniform in size and shape and their nuclei exhibit no mitotic figures which are the characteristic features of malignancy.

Age in-  
cidence.

Adenomata may develop at any age. They may be congenital also.

Treatment.

TREATMENT of Adenoma is complete extirpation as early as possible, to prevent its transformation into malignant tumours.

## SUMMARY.

### NON-MALIGNANT NEOPLASMS.

A. Hylomata.

B. Lepidomata.

Their differentiating features. The list of non-malignant tumours.

## A. HYLOMATA.

## I. LIPOMA.

## (a) Blastomata:—

## (i) Localized Subcutaneous Lipoma.

Treatment—operation.

## (ii) Deep Intermuscular Lipoma or Subfascial Lipoma.

## (iii) Parosteal Lipoma.

Treatment.

## (iv) Pericranial Lipoma.

Treatment.

## (v) Sub-serous Lipomata.

Treatment.

## (vi) Fatty Hernia of the Linea Alba.

Treatment.

## (vii) Sub-synovial Lipoma.

Lipoma Arborescens.

## (viii) Submucous Lipoma.

## (ix) Painful Lipoma of the foot or Tubby's Disease.

Nævo-Lipoma.

## (b) Blastomatoid, or Lipomatosis.

## (c) Mixtures and Transformations.

Fibromatous, Myxomatous and Sarcomatous Lipoma.

Xanthoma. Xanthoma Diabeticorum.

## II. FIBROMA.

## (i) Soft and

## (ii) Hard.

Epulis, Fibrous-polypus of the nose.

False-neuromata.

Blastomatoids.

## (i) Molluscum Fibrosum.

## (ii) Keloids or Cheloid.

Degenerations.

## III. CHONDROMA.

## (1) Echondroma. Solitary Type.

## (2) Enchondroma. Multiple Variety.

## (3) Chondroma of Teratomatous nature.

Blastomatoids. **Ecchondroses.****Chordoma.**

## IV. OSTEOMATA.

Blastomatoids. Osteoid and Hyperplastic processes. Metaplastic process.

## (i) Cancellous Osteomata.

This type is associated with growth and development. It is perhaps a blastomatoid process.

## (ii) Compact or Ivory Exostoses.

They develop in the cranium.

## V. MYXOMATA.

True Myxoma is perhaps unknown. Myxoma is perhaps a degeneration. Quick tendency to Malignancy. Treatment.

## VI. MYELOMA.

## VII. LYMPHOMA.

## VIII. MYOMATA.

## (i) Rhabdo-Myoma.

(ii) Fibro-Myoma, or Leiomyoma or Fibroids.  
The Uterine Fibromyoma.

## IX. ANGEIOMA.

## (i) Non-malignant Histoma.

## (ii) Malignant Cytoma.

## X. GLIOMA.

Other variations. Ependymoma. Gliomatosis. Diagnosis. Treatment.

## XI. NEUROMATA.

Many are blastomatoids.

## (1) Neuroblastomata.

## (2) Glanglioneuròmata.

## (3) Paragangliomata.

Carotid Tumours. Clinical Varieties.

## A. True Neuromata.

## B. False Neuromata.

## B. LEPIDOMATA.

## I. ENDOTHELIOMATA. (Non-malignant).

Two groups of Angeiomata.

## I. HÆMANGEIOMA.

Three Types.

## (i) Capillary or Simple Nævus.

Three clinical varieties of Capillary Nævus.

## (a) Mother's Mark or Simple Nævus.

(b) Portwine stain or Telangiectases and  
Nævus unius lateris.

## (c) Spider Nævus or Nævus Araneus.

## (ii) Cavernous Angeioma, and Hæmangeioma

Simplex.

Visceral Form in the Liver.

(iii) Plexiform Angeiomata. These are really

Aneurysms.

## 2. LYMPHANGEIOMATA.

Three groups, *viz.*,

(i) The capillary Lymphangeiomata.

Nævo-Lipoma.

(ii) Cavernous Lymphangeioma.

Macroglossia and Macrocheilia.

(iii) Lymphangiectasis.

(a) Simple.

(b) Cystic.

Cystic Hygroma.

## 3. SEROUS ENDOTHELIOMATA.

(1) Cerebral Membrane.

(i) Meningioma.

(ii) Psammoma.

(2) Pleural.

(3) Capillary vessels.

Psammomata. Endothelioma in the Pleura.

(i) Potato Tumour.

(ii) Perithelioma.

(iii) Mixed Parotid Tumours.

(iv) Giant-celled Tumours of the Tendon

Sheaths.

## II. EPITHELIAL TISSUE TUMOURS.

A. PAPILLOMATA. The structure of a Wart.

(1) Cutaneous Papilloma.

(i) Hard Warts.

(ii) Soft Warts.

(2) Mucous, or Moist Papilloma.

(i) Villous Papilloma.

(ii) Soft Papilloma.

(3) Intra-cystic Papilloma.

Blastomatoids of Epithelium. Callosity. Keratoma. Parafin or Billharzia Irritations. Condylomata and Coccidiosis.

B. ADENOMA. Fibro-Adenoma or better Adenofibroma. Two Types.

(i) Hard or Peri-canalicular.

(ii) Soft or Intra-canalicular or Cabbage-like.

Cyst-adenoma.

According to the histological character of the cells it is called.

(1) Columnar-celled Adenoma.

(2) Spheroidal-celled Adenoma.

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## CHAPTER IV.

### MALIGNANT NEOPLASM.

Before we proceed to study **Malignant Neoplasms** we must understand what we mean by the term **Malignancy**. Malignant neoplasms.

By the term malignant we clinically mean whatever ultimately ends in a fatal termination of the host. But this effect on the host may be due to indirect cause of mechanical nature, such as death produced by obstruction of the bowels caused by the mechanical pressure on the bowels by a non-malignant ovarian or uterine tumour. The fatal result in the latter circumstances is a mere accident and not due to any pathological property inducing it. Pathologically, by malignancy we mean certain distinct features and properties which although invariably turn the issue fatal as an ultimate effect on the host they do so in respect to these properties and not to the ultimate fate of the host in the shape of his death. That is to say, malignancy indicates certain cachectic or pathological effects on the host which is the criterion of the type of a malignant tumour. And therefore there are certain accepted **signs of malignancy** by which we clinically and pathologically describe a tumour as **malignant**. These are as follows:— Malignancy clinical and pathological.

#### SEVEN SIGNS OF MALIGNANCY.

Seven  
Signs of  
Malignancy.

- (1) Rapidity of **growth**.
- (2) Power of **autonomous** growth of **continuous** nature.
- (3) **Metastases** and **dissemination**.
- (4) Tendency to central **degenerations**.
- (5) **Ulcerations**.
- (6) **Cachexia** and **anæmia**.

(7) **Embryonic** character of cell.

Many of the above signs and some other clinical signs such as local recurrence after removal, may be misleading; as even a non-malignant tumour may recur at the neighbourhood of the site of the operation if a little portion of it is left behind. On the other hand the operation being undertaken in time, if we realize our situation as a thorough surgeon and remember the area of permeation while excising a cancer, there should not be any recurrence.

Character-  
istic patho-  
logical  
features of  
malignancy.

The characteristic pathological features of Malignancy by which we determine them to be so are the following :—

(i) Forma-  
tion.

(i) Their **formation** is always incomplete. They resemble no normal structure, organ, tissue, or cell.

(ii) Pro-  
gress of  
growth.

(ii) Their **progress of growth** is generally rapid. At the start they are usually single, rarely multiple. Malignant tumours are as a rule painless in its earlier stage, and since they give rise to no symptoms at the beginning it is impossible to guess accurately, the duration of the majority of the internal growths.

(iii) His-  
tory of in-  
cidence.

(iii) There is generally a **history of incidence** in the shape of irritation or chronic inflammation.

(iv) Age.

(iv) They usually grow after the adult **age**. But usually Sarcoma develops at the first half of life, Carcinoma develops after 30 years of age.

(v) Cap-  
sule.

(v) They are devoid of fibrous **capsule**.

(vi) Vege-  
tative or  
embryonic  
character  
of cells.

(vi) The cells are generally **atypical**, or aberrant in type, or **embryonic**. They deviate very widely from the normal histology of the affected region and therefore structurally and functionally they differ from the tissues or organs from which they develop. This deviation from the normal histological structure is called Anaplasia. The general rule is **the greater the degree of anaplasia the more the malignancy**. Both the **structure** of the cells as well as their **arrangements** and in some instances the distinctive **appearance** of the cells

and their highly specialized **function** are lost, *e.g.*, Non-malignant Adenoma of the breast approaches the structure of the normal mammary gland, but adenocarcinoma remains far from it; so also their prickles fail to reproduce their prickles and degenerate into simple forms. Sarcoma of the small round-celled type is the most malignant sarcoma, although the cells look very much like, only a little bigger than, the round cells of the blood. At the other side of the scale on the other hand some connective tissue tumours develop into perfectly **adult** type of cells and are entirely non-malignant; *e.g.*, in a Fibroma the parenchyma cells in it develop fully to an **ordinary normal histological adult** fibre, and for this feature it is classed as non-malignant; but as an intermediate example, in a Fibro-sarcoma although the parenchyma cells exhibit a **tendency** to become organized into a tissue which bears a **close resemblance** to the normal histological connective tissue, and develop **almost** into well-formed fibrous tissue but **not wholly so**, are malignant; because they do not develop **wholly so**. They are therefore a little less malignant than a round-celled Sarcoma. In a tumour like Sarcoma the cells do not develop beyond the embryonic stage, in a Fibro-Sarcoma they go a little beyond the embryonic stage, but **not up to** the exact normal type, but in a Fibroma on the other hand the tissue succeeds to reach the normal adult standard, and therefore not at all malignant. Therefore the degree of malignancy varies **inversely** to the success of development into adult standard.

(vii) They manifest very marked presence of (vii) Mitotic figures in the nuclei of cells indicating rapid growth.

(viii) They exhibit a characteristic feature of (viii) Infiltration. **diffuse infiltration** both local and general. They increase only to some extent by growth and expansion, but mainly do so by infiltration of the neighbouring tissues.

(ix) **Metastatic growth.**

(ix) **Metastatic growth** is always prominent, and when they are situate on the skin, they lead to ulceration.

(x) **Formation of Blood Vessels.**

(x) **Formation of blood vessels** is always incomplete. They may sometimes consist of thin-walled vessels but usually little formation of vessel wall is observed. Generally they are mere interspaces or interstices with little, or no vessel wall.

(xi) **Anæmia, Cachexia and Death.**

(xi) There is characteristic **cachexia**, or signs of general toxæmia, sepsis, infiltration in the glands, etc., finally ending the scene in **death**.

(xii) **Recurrence.**

(xii) They are generally always followed by **recurrence after removal**.

(xiii) **Increase of antitryptic power**, and lipoclastic action of the serum, are other evidences of malignancy.

## DISSEMINATION AND METASTASES.

**Dissemination and Metastases.**

Dissemination means the spread of the growth at the neighbourhood or anywhere of the affected person. Metastases mean secondary growths in the shape of colonies. A non-malignant tumour grows only by **expansion** of its own body as a toy balloon would expand on blowing. No part of it is separated from the tumour to grow elsewhere or carried by the circulation, its component parts being bagged in a capsule.

A malignant tumour on the other hand grows by **extension**, and has no bag or capsule and therefore every part of it is free to separate or wander out or extend as it would like utilizing any opportunity it may encounter to avail of. Such opportunities to enable a malignant tumour to spread are the following:—

**Methods of spread.**

1. **Infiltration** or penetration *via* tissue lymphatic spaces and interstices.
2. **Permeation** or penetration *via* the lymphatics.

3. Transplantation.
4. Embolism.

### 1. INFILTRATION.

1. Infiltration.

**Infiltration** consists of direct encroachment upon the tissue lymphatic spaces and interstices without any respect for the neighbouring tissues. The cells directly penetrate into any interspace or chink they can get in. Infiltration is the **earliest** disseminative process, and is best observed at the microscopic growing edge, within six inches from the visible margin of the tumour. It is a comparatively slower process, and is often interrupted as the tissues resist the encroachment. In this process the cells extend outwards between the planes of fascia, fat cells, fibrous bundles, muscle fibres, or wherever it can penetrate and consequently the tumour becomes very distorted and irregular in form. In the course of this extension the cells of the growing edge may encounter a vein or a duct or any large vessel to be carried away as emboli.

But in comparison to the next process of dissemination called Permeation, Infiltration is relatively a less dangerous process.

### 2. PERMEATION.

2. Permeation.

**Permeation**, or more correctly it should be described as Permeation of the Lymphatics.—By this process of spread, is meant the gradual but comparatively rapid method of extension of the outlying processes of the tumour in the form of solid plugs or columns of the tumour cells into, along, and always limited to, the lumen of the lymph vessels, which are more or less open tracks. The lymphatics so affected do not exceed more than 40—50  $\mu$  in diameter.

When such a process of extension takes place any or all of the four following things may happen; *viz.*:—

(a) Inflammatory perilymphatic reaction at the surrounding area of the lymphatic vessels around them which according to the methods of all reactive changes is evidenced by an accumulation of reactive and reparative cells such as the lymphocytes and fibro-blasts. This reparative attempt may be so successful as to convert the vessel into a small mass of fibrous tissue, and thus may very successfully obliterate the vessel and choke and kill the tumour cells finally effecting a cure.

(b) The vessel, through which the column of tumour cells makes its way like a finger into a tightly fitting rubber gloves, may be distended and as a result of such distension thus produced its wall becomes stretched and may finally burst; thus changing its method into Infiltration as a means for further subsequent spread. In this circumstance the inflammatory perilymphatic reactive process fails to arrest the spread.

(c) Whatever may or may not happen as described under the previous paras, the distal end or the growing tip of the plug may still escape the process of choking or ejection from the vessel by bursting, and proceed with the extension of the growth by further permeation of the same lymphatics, their branches, and the glands. So that it may appear as a solitary nodule at a distance from, and apparently unconnected with the primary tumour.

(d) While thus proceeding from a smaller to a bigger channel any part of the tip of the tumour column may get detached and float on as an **embolus**, and be arrested somewhere at a more convenient place, to proliferate into a secondary isolated nodule by metastasis.

Microscopic growing  
Edge.  
Extent and  
space of  
permeation.

Now **clinically**, the most important point is the **extent up to which this permeation takes place** and the tissue where it takes place. We cannot by naked eye examination actually determine the spot where the tip of the growing edge extends and clinically this margin or zone is inappreciable; and therefore this growing

edge may be described as **microscopic growing edge**. It has been found by all observers that this microscopic growing edge forms a small circle immediately around the growth and extends up to a diameter of roughly 2 ft., situated only in the **deep fascia** avoiding the smaller vessels and the larger trunks where the force of circulating stream is sufficient to sweep the protruding cells away as emboli, extending only through those with a lumen of 40—50  $\mu$  in diameter.

We should therefore in all our operations or radium therapy consider this centrifugal extension and always bear in mind three distinctive zones, around the central point of infection, *viz.* :—

(a) A circle of secondary nodules each of which may be isolated, or all of them confluent or contiguous to each other, the intermediate permeated space or zone being destroyed.

(b) External or peripheral to the above, a zone of Perilymphatic Fibrosis.

(c) The Microscopic Growing Edge.

An operation for the cure of cancer can only be possible and successful when the whole area consisting of the three zones is thoroughly removed.

### 3. TRANSPLANTATION.

#### 3. Trans-plantation

Dissemination taking place without the help of lymph spaces or blood vessels, but where the seeds transfer and grow at a new place is called Transplantation. Transplantation may take place under the following circumstances. *Viz.* :—

(a) By 'Kissing contact' or Transplantation by Apposition. This form of Transplantation is not very frequent. It takes place where a surface is in actual direct contact with a malignant growth, *e.g.*, a growth on an affected labium transmitted to the other, or a cancer of the cervix uteri to the wall of the vagina.

Unless there happens to have some injury or abrasion on the unaffected surface the transference of the tumour cells is not probable on a normal epithelium.

(b) Transplantation by Inoculation. If in the course of removal or exploration the tumour cells are left behind on an injured surface they may behave as grafts to grow on the raw surface, *e.g.*, an exploration done on an enlarged liver for diagnosis, if affected by carcinoma, may be the cause of producing cancer on the surface of the abdomen at the site of the puncture. During operation of excision of the cancerous breast the tumour cells may be easily transplanted on the incision wound unless the flaps are carefully protected.

(c) Transcœlomic and Trans-serous Transplantation. This method of transplantation occurs should a malignant growth burst through the lining membrane into a serous cavity, such as the peritoneum or the pleura. This form of transplantation may also occur as an ultimate end of permeation, *e.g.*, there are numerous fine anastomoses which pierce the parietes, and connect the lymphatic plexus of the deep fascia with the sub-endothelial lymphatic plexuses of the pleura and peritoneum, and the mediastinal and portal glands. Permeation along these lymphatics, *e.g.*, a breast cancer is the main source of visceral metastases. As soon as the subserous plexus is reached the cancer cells may easily erode the overlying endothelial layer and escape into the peritoneum. Thus breast cancer may be seen with metastases on the ovary, as the *gravity* and *muscular movement* help the free cancer cells to migrate diffusely inside the peritoneal and pelvic cavity. It may be remembered that in the epigastric angle it is only the linea alba constituted of a single layer of fibrous tissue which separate the deep fascia of the parietes from the subperitoneal fat, and the distance from the mammary cancer circumference to the epigastric angle is **only one inch**, a space which may soon be traversed.

This extension is clinically described as **Epigastric Invasion of the Abdomen.**

#### 4. EMBOLISM.

4. Embolism.

4. **Embolism**—This method of metastasis need not be detailed here. Embolism is the method which was believed to be the only way of dissemination in the former days. In malignant tumours of connective tissue origin the parenchyma cells are in close contact with blood vessels, and therefore we find sarcoma is more easily disseminated *via* blood vessels to produce metastases. In infection we found the first set of colonies settle in the lungs which is the first port of arrest, but this is far true in malignant dissemination, and lungs are often found apparently clinically free from such deposits when other organs are affected. As a matter of fact they do occur but become surrounded by thrombosis which successfully encapsules or destroys them. *Retrograde Embolism, Tissue Predilection* are other theories to explain various other anomalies of metastases. Embolism occurs more frequently than so much of metastases, and it is doubtful whether all emboli retain their power of metastasis.

Metastasis is affected in various other ways, and like infection it exhibits an extraordinary degree of variation according to the following conditions. *Viz. :—*

- (1) Tissue Predilection.
- (2) Size.
- (3) Number.
- (4) Time of appearance.
- (5) In the different classes of Neoplasms.
- (6) In the different sites of the body in similar tumours.
- (7) In clinical importance.
- (8) Its comparative development in relation to the primary growth. Occasionally the secondary growths outbid the primary one in clinical picture.

The modes of metastases and extension sometimes clinically help us to ascertain the extent, and to diagnose the kind of malignant growths, *e.g.*, carcinomata spread by means of the lymphatics, and hence secondary growths are very common in the lymphatics. In sarcoma, which disseminates *via* the blood stream, the viscera are more affected than the lymphatic glands. On the other hand in glandular or adenomatous carcinoma the viscera as well as the lymphatic glands are **both** affected. In squamous epithelioma it is only the lymphatics which are affected, the viscera generally escape.

The degree of malignancy and their variation in secondary growths.

The local symptoms and general signs of malignancy **proportionately vary** in different tumours according to the **degree** of malignancy, and especial peculiarity of some particular tumours. In some tumours the local lesions and in others the general symptoms, more or less predominate; for instance, in Melanoma, the primary growth appears to be insignificant, but the secondary disseminations in the viscera may be very extensive and immediately fatal. On the other hand a rodent ulcer will never produce any visceral or other secondary growths, but the local destruction is very slow and may be horribly mutilating. The latter is non-malignant in the opinion of recent observers. Roughly speaking cancer has never proved local at any stage, or not to be general at every stage.

The consistence of a malignant growth varies with malignancy.

**The consistence** of a malignant growth varies with malignancy. That is to say, the more malignant and rapidly growing a neoplasm is, the less becomes the chance for the stroma to proliferate. The slower the growth the more abundant is the formation of the stroma. This comparative absence in one case or abundance of stroma in the other, makes the neoplasm **hard** as in scirrhus cancer, or **soft** as encephaloid cancer; and clinically we describe them as such according to consistence.

## GEOGRAPHICAL DISTRIBUTION.

## THE INCIDENCE OF CANCER IN INDIA.

It is interesting to observe that the neoplastic growths have some peculiar geographical distribution. It is believed to be more common in the west than in the east. Even in the west the mortality in cancer has increased more than twice in the space of two generations. Probably few families of ordinary size escape cancer in three consecutive generations. One person in seven over the age of 30, dies of cancer in England.

The Incidence of Cancer.

In India the Himalayan tracts are definitely more favourite soils for malignant neoplasms than the plains. In Nepal and Kashmir cancer is as common as in England.

Incidence of benign tumours are more or less equally distributed all over the world.

Malignant transformation of ulcers, is not as common in the east, or warmer regions as it is in colder countries.

## AGE INCIDENCE

Neoplasms, particularly those of malignant type are seen in younger patients in the tropics than the average age at which they occur in the colder countries. In India "economic distress and nutritional defects of the indigenous population have been referred to as direct or indirect causal agents."

Age Incidence.

In this connection, dirty habits of the Himalayan people may to a certain extent account for many malignant neoplasms at an earlier age. In this earlier age tumours, if the age factor is seriously allowed for, squamous-celled cancers are much more common in Bengal. Cancer of the tongue and buccal region and penis in the males, and that of the cervix and liver in females, are common amongst Bengali Hindus at a comparatively younger age. Of all the known factors age

is by far the most constant and significant one. If we say cancer is unquestionably a **disease of age**, and the cancerous process is a **function of age**, we ought to substitute the term **senility** for age in India. It is not the question of the number of years a person has lived, but it is always a question of age **conditions**; and in Bengal we ought to say cancer is a function of pre-senility. The age factor is in truth dependent upon age retrogression. It may be a question of local senility rather than general senescence. It is the degree of senility which is the true measure of the proneness to cancer, that is to say, the more senile the patient the more susceptible is he to cancerous growths. He need not necessarily be old in age. On the other hand in advanced senile atrophic conditions cancer hardly develops. Tissues and organs which have **started** to decline and slide down hill to retrogression are most prone to develop cancer.

### SEX INCIDENCE.

Sex Incidence.

The general idea in India is that female sex is prone to cancer, but the only part played by sex in the incidence of cancer is the determination of its relative site incidence as a help to correct diagnosis, *e.g.*, in the female the cervix, uterus, ovary and the mammary glands are the most frequent seats of malignant tumours; whereas in the male the buccal mucosa, tongue, stomach, rectum, and penis, are the commonest sites. Amongst unmarried women and amongst widows cancer of the breast and ovary are a little more frequent than amongst married women. Cancer of the cervix on the other hand is more prevalent among married women.

Site Incidence.

### SITE INCIDENCE.

Gastro-intestinal tract.

The feature of site incidence in India is a little different from what is found in the west. Gastro-

intestinal cancers exhibit greater preponderance in England than in India, although chronic dyspepsia, amœbiasis, diarrhœa, dysentery, hook-worm, typhoid, etc., are more commonly met with in India. True ulcers in the stomach, papillomata, adenomata, etc., are uncommon, and even in some districts affected by gastro-intestinal infections where a few cases are met with they do not seem to lead to malignancy to any great extent. Rectal adenoma and piles become easily cancerous. Primary cancer of the liver is common in Bengal. It is as common amongst orthodox Hindus, as the staunch Mahomedans. Amongst women, whether meat-eaters as the Mahomedan women, or orthodox Brahmin widows who are strictly vegetarians, cancer of the liver is equally seen. Cancer of the liver is seen at all ages from 25 to 60. It is often preceded by amœbiasis or cirrhotic changes in that organ. Possibly either the parasites in the living condition, or the toxin of dead parasites produces some chronic irritation of the liver tissue resulting in cell proliferation, and hyperplasia of blastomatoid nature which ultimately end in tumour formation. In Europe primary cancer of the liver is very rare. In the west, cancer of the liver is usually secondary and it starts in the intrahepatic bile ducts and bile passages from cancer of the colon and upper part of the rectum but in the Tropics it originates in the liver cells. Several varieties of liver-cell carcinomata are described in the west also.

It is interesting to observe that although spleen receives its blood supply from the same source as the liver, cancer of the spleen is very rare.

We have already stated that the occurrence of cancer is influenced or excited by antecedent conditions which are generally of the nature of constant irritation or chronic inflammation, infective or otherwise. Hutchison many years ago described this antecedent condition as "precancerous condition". If we could educate the public to be acquainted with this precedent

Liver.

Spleen.

Education  
of the  
public.

Warning  
of the pre-  
cancerous  
conditions.

phase, in certain instances cancer may be prevented. These "precancerous conditions" in the digestive tract excluding the buccal cavity are the following; in the œsophagus, stricture from injury, caustics, etc.; in the stomach, peptic ulcers; in the liver, amœbiasis and other parasitical inflammatory hepatitis, chronic cholecystitis, gall-stones, etc.; in the intestines, chronic inflammation, adenomata and diarrhœa; in the pancreas, chronic pancreatitis and pancreatic calculi; in the rectum, adenomata, fissures, hæmorrhoids, chronic symptomatic diarrhœa, etc.

Buccal  
Mucosa.

Malignant growths of the buccal mucosa, and the face, the jaws, the tongue, are frequent in India. They are generally of a virulent type. Betel mixed with lime, raw tobacco leaves, and other substances as *soorti*, *jardah*, musk, constantly chewed and kept in the form of a bolus or a mass under the tongue, or between the gum and the cheek, are the fruitful causes of exciting malignant growths. Pre-existing leukoplakia from syphilis excites the progress. Sometimes non-malignant tumours also develop on the tongue, especially of young women (*Vide* photo plate No. XXXVII which may attain enormous size. The "precancerous conditions" of the tongue are leukoplakia, jagged teeth, wounds, scars, etc.

Uterus and  
cervix and  
breast.

Cancer of the cervix-uteri is comparatively common amongst married women, and sometimes amongst young women. Cancer of the uterus is also fairly common amongst women after climacteric. It is curious that uterine and mammary cancers are fewer in India amongst lower class of people than upper classes; at least they are not as commonly met with as in any other countries. Lacerations and erosions of the cervix, endometritis, repeated pregnancies, are the respective precancerous conditions of the cervix, uterus, and ovaries. Chronic mastitis and chronic eczema of the nipple, repeated knocks received on painful parts of the breast, may induce cancer of the breast.





SQUAMOUS CANCER, ULCERATIVE.

Cancer of the penis is a frequent occurrence amongst Penis, etc. Indians. Syphilitic history has been determined in more than sixty per cent. of cases. Circumcision is not proved to be preventive. *Vide* photo plate No. XLV.

Malignant growths in the skin is not uncommon in Skin. the Tropics. It is generally associated with some parasitical infection or irritation from ulcers left after burns, chemical irritants, lubricating oils, wounds, and exposure to heat, (*vide* photo plate No. XLII. The most frequent "pre-cancerous" conditions of the skin are, chronic dermatitis from exposure to X-rays, pigmented nævi or moles, lupus vulgaris, chronic scaly or fissured lip, keloids, *vide* photo plate No. XXXIII ulcers from constant irritations, etc. Melanoma are not met with in the coloured nations, but amongst the white races it is easily excited to malignancy.

### CARCINOSIS.

In this connection of local site incidence, it may be added that, although in most cases a local "precancerous" condition determining the disease and registering it at a particular local site, is found, in most cases there exists also a **systemic condition**, described as **carcinosis** which is the essential morbid basis of cancer, and which would represent both; *viz.*, (a) a general or systemic or predisposing **cancerous**, condition, and (b) a **local** or inflammatory or exciting **precancerous** state. "Carcinosis" is influenced by metabolic and nutritional factors, and "precancerous" condition may be produced by internal as well as external factors, *e.g.*, mild low-grade chronic inflammations resulting in ulceration, hyperplasia, or fibroblastic blastomatoid process; all these latter factors amount to a diminished arterial supply, a lessened cell resistance and vitality, which mean a preparation of a suitable soil, for the neoplastic growth.

"Carcinosis" and "precancerous" condition.

(a) A general factor, and  
(b) A local factor.

A preparation of a soil for the growth.

## TISSUE PREDILECTION IN METASTASES.

Tissue  
predilec-  
tion in  
secondary  
growths.

We have explained the process of Dissemination and Metastasis. But so far as the different sites where secondary growths appear there is some peculiar tendency to particular site selection. This peculiarity cannot be always explained by any of the different processes of dissemination. The different positions in which secondary foci develop in various tumours show a remarkable tendency to appear according to their own choice of tissues or organs.

Metastasis is very rare in the spleen, whereas liver is soon riddled with it, from even a small primary focus in the colon; and although lymphatics are commonly affected it is strange how extraordinary number of metastases are found in the skull bones in the cancer of the prostate whilst only a few lymphatic glands may be affected in the lumbar region. It is not clear why in melanomata of the eye and skin, the liver should be so early and diffusely affected. Of the other organs the brain, kidney and pancreas are less frequently involved. Metastases are very rare in skeletal muscles. We are thus faced with the fact that certain organs and tissues appear to offer an unusually favourable soil for the metastases of some particular kind of tumours.

The malign-  
ant Atypi-  
cal Cyto-  
mata :—  
I. Epi-  
blastic  
Cells.

The malignant or **Atypical** Cytomata we are to deal with are the following :—

### I. EPIBLASTIC CELLS.—

#### (1) Carcinomata.

(i) Squamous-celled Carcinoma.

(ii) Spheroidal-celled, or Carcinoma Simplex.

(iii) Columnar-celled, or Adeno-carcinoma.

#### (2) Neurocytomata.

II. Meso-  
blastic  
Cells.

### II. MESOBLASTIC CELLS.—

#### (1) Endothelial.

(i) Endotheliomata.

(2) Desmocytomata (Sarcomata).

(i) Round-celled Sarcoma.

(ii) Spindle-celled Sarcoma.

(iii) Giant-celled Sarcoma.

(3) Lymphocytomata (Lymphoid cells).

(4) Myocytomata (Muscle cells).

(5) Blastocytomata (Mixture of Indifferent cells).

### III. PIGMENTED TUMOURS.

III. Pigmented Tumours.

### IV. HYPERNEPHROMATA.

IV. Hypernephromata.

### V. TERATOMATA, or ORGANOMATA or EMBRYOMA.

V. Teratomata  
Organo-  
mata.

## I. MALIGNANT EPITHELIAL TUMOURS.

### (1) CARCINOMA.

Much spade work has been done regarding Carcinoma in the previous chapters. All questions and investigations in relation to tumours in general, the study of their features, and the methods of their treatment, the attempts at radical cure are at the present day of surgery more or less focussed to **carcinoma**; and the problem yet remains as difficult, and as unsolved to-day as it was from the time humanity are trying to solve the problem of their ailments. Present day surgery is getting tired of the unceasing use of its knife on longstanding cancer, but it is getting more vigorous in completely extirpating the mass with its possible lymph drainage in early cases.

To proceed now to a detailed study of the various types of carcinoma, we come across with tumours derived from all the various epithelial tissues of the body which manifest a correspondingly wide variations in their structures.

The main sources from which epithelial proliferation of malignant or carcinomatous nature may originate are the following :—

- (a) From the lining epithelium.
- (b) From the glandular epithelium.
- (c) From epithelial rests.
- (d) From cells of non-malignant epithelial tumours, *e.g.*, the papillomata and adenomata.

Essential  
characters  
of carcino-  
matous  
growth.

The essential character of a carcinomatous growth consists in the following features; *viz.* :—

(i) Lost-  
control.

(i) Unlimited multiplication. We have already explained the theory of **lost balance** and **tissue tension**, *vide* Chapter II of this Volume. The epithelial tissues in our system are constantly changing and renewing under some control in our system. This activity of epithelial proliferation is looked upon as of a defensive nature in our system. This control seems to be entirely lost on the revolutionary, aggressive, encroaching, and growing cells of a carcinoma. *Vide* p. 78.

(ii) Atypical  
and  
embryonic.

(ii) But these growing cells show little resemblance to normal tissues or glands or structures. They are absolutely **atypical** and never attain the adult type, remaining *always* **embryonic**.

(iii) Ana-  
plastic.

(iii) They lose all their specialized characters, and revert to simple masses of protoplasm, and thus become **anaplastic**. The more the malignancy the more anaplastic they become.

(iv) They  
only  
divide.

(iv) They only know one thing, and adopt one character, and that is of **division**, **sub-division** and **growth**.

(v) Absence  
of unifor-  
mity  
amongst  
themselves.

(v) Mutual relationship is lost and they thus greatly differ amongst themselves in the form of division, character of nuclei, and their own sizes. The division is mostly **mitotic**, **irregular**, **tripolar** or **multilocular**. Divide and divide is their constant cry.

(vi) They keep no relationship with their neighbours, and they **never** remain **limited** to their own sphere.

(vi) Absence of relationship with their neighbouring tissues.

(vii) **Aggression** and **encroachment** are their constant habits. To advance and proceed onward and **annex** other's dominions is their constant **policy**. **Infiltration** is their method, which is accomplished in the way of peaceful penetration into others' domains, at first like a guest but finally assuming occupation and rights of the host, till they are in final possession of the latter's hearth and home. This infiltration is effected by the epithelial cells penetrating into the tissue, taking the lines of least resistance and is usually done through the lymphatic clefts, and then through the lumen of the lymphatic vessels when the process of extension is described as **lymphatic permeation**. When such conditions happen, no visible line of demarcation remains to help us to distinguish between the epithelial and the connective tissues, and the two appears to be inextricably blended.

(vii) Aggression and Encroachment upon other's provinces.

(viii) Around the growth marked **reactionary** phase is observed in the connective tissues almost similar in nature to inflammatory reaction. Many small round cells, plasma cells, and polymorphonuclear leucocytes, appear at the scene. Infiltration of these reactive and reparative cells lead to organization and formation of **stroma**, around the epithelial columns. According to the rapidity and acuteness, or chronicity and slowness of this organization, the **density** and **vascularity** of the stroma vary. In **acute** or rapidly growing cases the stroma becomes **vascular** and **cellular** and comparatively **small**, and as the tumour appears like brain substance, is described as **encephaloid**. In **chronic** or slowly growing tumours the stroma becomes **fibro-cicatricial** containing **few blood vessels**, and as its consistence becomes hard or stone-like it is described as **scirrhus**. Polymorphs become abundant in the cases where ulceration has occurred, in which circumstance the inflammatory

Encephaloid and Scirrhus.

manifestations become more evident by the association of pyogenic bacteria in the body of the neoplasm.

(ix) The neighbouring **glands** are involved sooner or later, in some cases very early, and their enlargement is an important diagnostic sign. If the glands are involved by infective inflammation or ulceration, the toxin absorbed by them makes them comparatively bigger in volume than those carcinomatous glands which are not complicated by any infection or ulceration.

(x) When the nerves are implicated and dragged in the substance of the harder forms of cancer the tumour becomes tender, associated with a considerable degree of pain of neuralgic character. Otherwise cancerous growths are not tender.

Develop-  
ment and  
termina-  
tions.

**Development** of the growth and its **termination** usually occur as follows. At first it appears as a **primary** tumour which is almost invariably **single**. Growth then continues involving the neighbouring tissues which are **infiltrated**, **destroyed**, and **incorporated** into the substance of the tumour surrounded by a zone of inflammatory processes. **Extension** then proceeds on all sides towards the surface, and to the deeper structures. If it can reach the surface **ulceration** takes place, which coming in communication with the exterior is infected by pyogenic micro-organisms, and is therefore affected by a very foul smelling area of **inflammation** bounded by a zone of acute irritative reactions. The extension *via* the lymphatics affects the **glands**, where **colonization** occurs which behaves in the same way as the primary growth. Gradually as dissemination takes place as described before, the affection behaves like chronic pyæmia, and the **toxin** as well as the **tumour cells** ultimately get into the the general **circulation**, by which they affect the viscera, *e.g.*, the lungs, liver, brain, and bones. It is this **embolic** process which produces grave and fatal **toxæmia** which is always followed by **death**.

The tumour cells which apparently behave like an inflammatory focus can be injected and inoculated and

in this way reproduced in the same way as any granulomatous nodule, such as a tubercle, or any septic substance, or bacteria, producing localized inflammation. But there is some characteristic difference between the two processes: *viz.*, (i) in infection the tissue cells of the inoculating mass die out quickly but the micro-organisms continue to live, and reproduce the characteristic changes in the tissues of the animal inoculated. (ii) In infection inoculation can be done in **any animal** irrespective of serological difference. Whereas in the case of carcinoma, (i) the living neoplastic **cells** or **tissues, live** and **multiply**, and the further process of its existence is one of **transplantation** and not of inoculation. If by this method of production any truth of the parasitic theory of carcinoma be attempted to be proved it may be noted that the parasites are possibly contained **within** the carcinomatous cells. (ii) Grafted cancer cells do not develop in the animals other than that of the same species from which the graft is taken. Mouse and rat are the only animals in which the experiments are successfully propagated. Koch's canons of infection are not satisfied in these experiments by inoculation, and therefore these experiments are followed by very poor success.

But there are some marked features which resemble infection; *viz.*: immunity and natural cure, are sometimes observed in carcinoma although such cases are **rare**. The very fact that inoculation is mostly unsuccessful admits the proposition that some amount of **natural** immunity does exist; and the experiments indicate that the conditions of this natural immunity must be present in the tissues or blood of the host that are favourable to the tumour. At the same time mice from different localities exhibit different degrees of susceptibility to the same tumour, serving corroborative evidence in the existence of natural immunity. To a certain degree, immunity can be acquired, may be proved by the fact that a mouse unsuccessfully inocu-

Similarity with infective processes.

Natural immunity.

Acquired immunity.

lated with tumour cells of low virulence behaves in a very refractory manner to the inoculation of tumours of great malignancy, although the latter operation may be effected successfully in normal mice having had no inoculation previously. Some tumours are highly virulent and their virulence increases by repeated inoculation and transmission through susceptible animals; in this respect the neoplasm behaving like some virulent infections.

**Natural  
cure.**

It has been observed that well-diagnosed cases which were given up as inoperable and hopeless ended in a spontaneous **natural cure**. This may be argued as instances that some kind of acquired immunity can be developed by the process of automanufacture of some substance produced in our system which can effect a cure, although we do not know what that substance is. We have described how to a certain degree automatic cure is effected by the reactive processes around permeated lymphatic vessels.

Physiologically we come across mainly of the following three types of epithelium, *viz.*:—

(1) **The Squamous Epithelium**,—is found on the:—

- (i) Epidermis or superficial part of the skin.
- (ii) Mucous membrane of the nasal orifice, tongue, mouth, pharynx and œsophagus.
- (iii) Conjunctival epithelium covering the cornea.
- (iv) Lining the vagina and the vaginal part of the cervix uteri.

In all the above situations the epithelium is of stratified nature, that is to say many layers are in superimposed position; but the superficial cells are as a rule of flattened or horny and **squamous** or scaly variety. In squamous variety the cells are flat having its two diameters long, and one very short, something like the tiles used to cover the roofs of buildings, or plaster the rooms such as public halls and lavatories.

(2) The next type of epithelium is the **columnar** form. In this one diameter is long and the two others are short resembling a brick standing on an edge. The deepest layer of cells in the stratified epithelium are columnar in nature. In the mouth and the skin the outline of the deeper cells is very irregular, which form processes by prolongations of the intercellular net work. They are called **prickle** cells.

(3) The third type of epithelium is the **cuboidal** or **spheroidal** form. In this form the three diameters are more or less equal, resembling something like a cubical box or a spherical ball equally flattened at all the six sides. These cells line the glandular structures such as the breast, the prostate, the kidney, etc.

The urinary bladder and the ureters are lined by typical **transitional epithelium**. This term is applied to epithelial cells which are found in two or three superimposed layers, the upper being composed of cells which are more or less flattened, but which never become squamous normally; and if they become so it must be understood that they are tending to carcinomatous condition.

The different varieties of Carcinoma met with, arising from the above, are the following:—

- (i) Squamous,—sometimes called only Epitheliomata.
- (ii) Spheroidal or Cubical, or cuboidal.
- (iii) Columnar.

The differentiation consists in the nature and appearance of the cells.

## CLINICAL VARIETIES.

Clinical  
varieties.

### A. Surface Type.—

- (1) Squamous Carcinoma, and,
- (2) Rodent Ulcer.

**B. Gland Type.—**

(1) Spheroidal or Cuboidal Carcinoma.

(2) Columnar Carcinoma or Adeno-carcinoma  
and **Paget's Nipple.**

The broad features by which a Sarcoma may be differentiated from a Carcinoma are the following:—

(i) In Carcinoma the connective tissue coat round each individual cell is absent, that is the cells are collected into groups separated by a stroma, although the stroma does not penetrate between the individual cells of which the group is composed in an alveolar form.

(ii) **Metastases** in Carcinoma are always present. In some Sarcomata metastasis may not occur.

(iii) The cancer cells are squamous, spheroidal or cuboidal, and columnar; or they may be of normal appearance. In Sarcoma it is round or spindle-shaped.

(iv) The cells in cancer arrange themselves in an alveolar form, or sometimes in masses, as stated above. But in sarcoma the stroma and parenchyma are intimately mingled together, each parenchyma cell being isolated from its neighbours by a delicate fibrillæ of stroma.

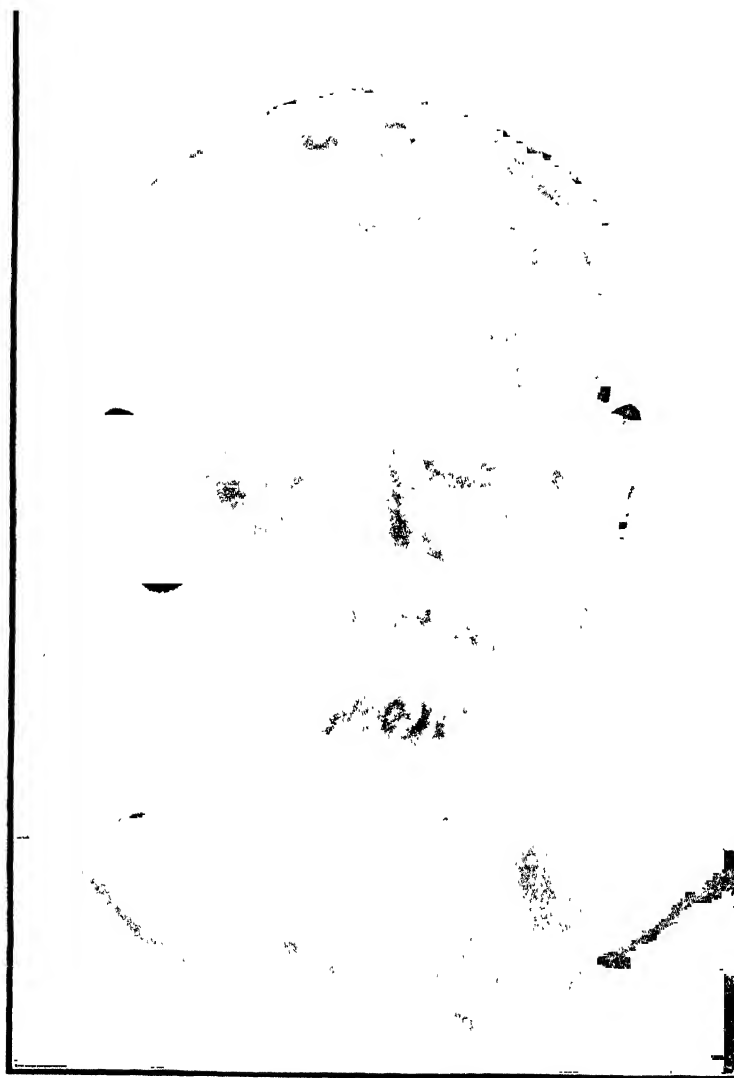
**A. Surface types.****A. SURFACE TYPES.****1. Squamous celled Carcinoma.****1. SQUAMOUS CELLED CARCINOMA.**

**Squamous Celled Carcinoma** is also described as Epithelioma, Squamous Epithelioma, Epithelial Cancer, etc. We have already explained our reason how it would be less confusing to the beginners, and pathologically as well as logically more correct to use the term Epithelioma for all non-malignant and malignant epiblastic and hypoblastic neoplasms that is any tumour derived from epithelium, as is employed by Ziegler.

**Sites.**

Squamous Carcinoma is a cancerous tumour growing from the surface tissue, that is the epithelium of the lining





SQUAMOUS CANCER OF ANTRUM OF HIGHMORE.

or **protective** type, such as the skin, or from those parts of the mucous membranes which are covered with **squamous** epithelium; *e.g.*, mouth, pharynx œsophagus, the lining of the pelvis of the kidney, ureter, bladder, vagina, and cervix uteri. These are the situations covered by squamous epithelium, except those of the bladder and ureter being transitional. *Vide* page 224.

AGE of onset.—Squamous Carcinoma usually appears in middle-aged and elderly people; occasionally it is seen in young adult life.

Age of onset.

### ÆTIOLOGY.—

**Irritation** of some continued nature: *viz.*; (i) **mechanical**, *e.g.*, a friction of a clay pipe over the lips, or bad or sharply broken tooth at the side of the tongue; (ii) **chemical** such as works with paraffin or tar, or the use of some contraceptives inside the vagina; (iii) **thermal**, *e.g.*, habit of swallowing very hot eatables and drinks, constant contact of a hot clay pipe; (iv) **physical**, such as the palki-staff over the shoulders of the bearers, (v) **electrical**, such as the continued exposure of X-rays on the hands, mouth and exposed surfaces of the radiologists, (vi) or other pathological conditions as a **scar** or **lupus** or ulcer, etc., are some of the exciting causes which may induce cancerous proliferations of the **surface** epithelium.

Ætiology.

**Clinical Types.**—In this connection the reader may usefully revise the pages on non-malignant epithelial tumours. In the early stage there may sometimes be little difference in clinical history and external appearance between the different types of non-malignant epithelial tumours such as papillomata and adenomata and malignant epithelial neoplasms, called Carcinomata.

Clinical types and their respective histological features.

Clinically a Squamous-celled Carcinoma may be looked upon as a Malignant Wart. A non-malignant wart is a Squamous Papilloma covered with flattened epithelium of squamous type, which grows outwards from

the surface; but a malignant growth of this nature grows underneath it than above the surface, burrows deeply into adjacent tissues, and being on the surface sooner or later is ulcerated, which having been exposed, is secondarily infected. The tumour consists of finger-like processes or large solid masses of cells in broad branching columns growing down below the level, which include various structures representing elements present in normal epithelium.

Normal epidermis consists of the following structures from above downwards. *Viz.* :—

1. Layer  
of Keratin.

(1) Stratum Corneum—a layer of cells with **keratin**, which is derived from the keratohyalin in the next layer; or a horny epithelium consisting of granules of a substance like beeswax.

2. Layer of  
Kerato-  
hyalin.

(2) Stratum Lucidum—a layer of cells with flattened nuclei in which eleidin derived from the next lower layer is changed into **kerato-hyalin**.

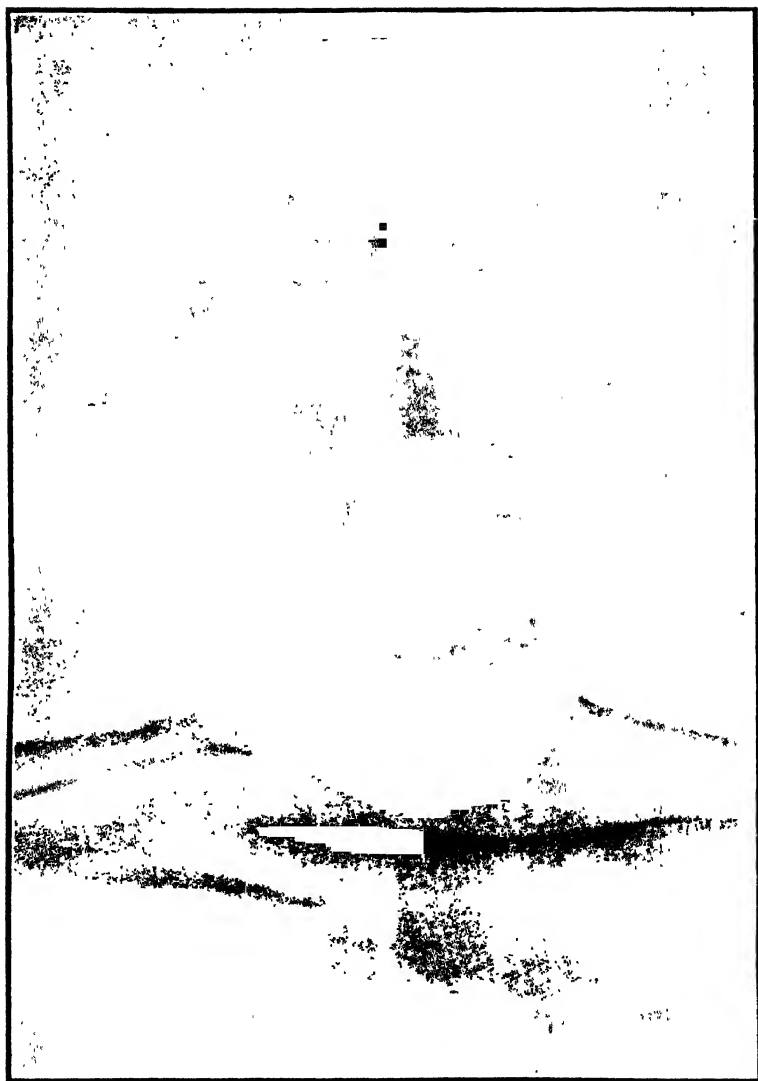
3. Layer of  
Eleidin  
formation.

(3) Stratum Granulosum—are two or three layers of cells of fusiform shape containing granules of **eleidin**, a substance which is an intermediate stage in the formation of **keratin**. This is the most important layer as it is here where transformation, from the protoplasmic cells of the lower strata called stratum mucosum to the horny or keratinized cells of the upper two layers, takes place. These two or three layers are important from pathological point of view to detect neoplastic transformation.

4. Layer of  
Prickle  
cells with  
Lymph  
Channels.  
Layer of  
Permea-  
tion and  
Pigments.

(4) Stratum Mucosum—consists of several layers of **spheroidal** or polyhedral cells containing soft, opaque, and granular substance the surfaces of which are beset with numerous short thorn-like processes interlacing with those of the neighbours forming bridges which give them a characteristic appearance and have led to their being especially called **prickle-cells**. Between these bridges are the minute **lymph-channels**; the most important layer in connection with lymphatic permeation in cancer. This permeation may be very easily demonstrated in the





SQUAMOUS CANCER, PAPILLOMA OF LIP.

Paget's disease of the nipple. Pigments in coloured races are especially distinct in this layer.

(5) Stratum Germinativum.—This consists of a layer of **columnar** cells with oblong nuclei. The cells stand on edge perpendicularly on a basement membrane attached to it. It must be remembered that when this basement membrane is encroached upon by any growth as occurs in all forms of carcinoma, it must be taken as malignant. So long as the growth is confined above this membrane it should be understood to be non-malignant. This is the layer where Rodent ulcer develops and stops short, without immediately encroaching upon the basement, and it is for this reason a rodent ulcer is described as non-malignant by the Continental authorities. In the belief that it grows from the basal cells of the epidermis they are called **basal-celled** Carcinoma. It is in this layer also that **nævus** cells containing pigments and **melanin** originate.

5. Layer of Multiplication and Pigments.

Layer of Nævus cells. Melanin of Melanoma.

**Microscopically**, in Squamous-celled carcinoma the diseased area as described above consists of broad branching **cancer columns** of large solid masses of epithelial cells ramifying like interlacing and irregular net-work, working deep in the subcutaneous tissues constituting the main **parenchyma**. The intervening meshes of this net-work ramification are occupied by fibro-cellular **stroma**. The true **cancer cells are derived from the prickles** containing **eleidin** granules in the process of formation, although in many cases, especially in the rapidly growing ones, it is difficult to find the prickles, unless especially sought for and that also at some isolated areas where they are distinctly evident. The multiplication in number and division of the cancer cells take place prominently nearest the stroma, and thus in the columns of the epithelial outgrowths differentiation of the cells into three *distinct* layers could be made out; *viz.*, nearest the stroma and in contact with it the cells resemble the basal layer or stratum germinativum of the normal skin, and are

Microscopical appearance. Cancer columns constituting the Parenchyma and stroma.

usually regular in nature; next to this normal looking cells, neoplastic cells are situated nearer them which are polygonal in shape, in the deepest layer the cells are oldest of all in which the degeneration is complete, and these become flattened and keratinized; thus forming in the centre a whorled arrangement which are described as **cell-nests** or **epithelial pearls**. It must be very carefully remembered that these **keratinized cell-nests** are distinguishing marks of Squamous-celled carcinoma, and if we come across with cell-nests anywhere else in any other organ and tissue where normally keratinized squamous cells do not exist we must at once conclude that it is a migrated metastasis secondary to some squamous celled carcinoma elsewhere on some surface. The diagnostic feature of this variety of tumour therefore are:—

- (i) Presence of cell-nests.
- (ii) Presence of prickle cells.
- (iii) Presence of cells containing **eleidin** granules.
- (iv) Presence of keratinization.

Cell-nests  
in the substance of the  
cancer  
columns.

It must be remembered and noted well always that it is in the chronic cases where **cell nests** are well formed in the substance of the cancer columns, distinctly exhibiting this arrangement into three layers in a very characteristic fashion. It is the older portions where the cells are concentrically arranged, becoming flattened and undergoing keratinization transformed from the **eleidin** granules which may be so complete that the **epithelial pearls** or **cell-nests** are formed. Cell-nests are not formed in acute cases. In carcinoma of the œsophagus and pharynx the keratinization may be rudimentary. In the antrum of Highmore also a type of atypical carcinoma of Squamous type is formed. *Vide* photo plate No. XLIII.

Epithelial  
pearls.

These **epithelial** columns have no particular shape, but form into irregular outgrowths or processes. On section these columns appear to be oval or round according to the point of section taken out from the main mass at the





NODULAR CANCER OF PENIS.

growing margin. In the marginal zone round-celled infiltration and collections of plasma cells may be observed.

Difficulties sometimes arise in the minds of beginners how the keratinized cell-nests appear so deep. It may be easier to imagine that if we dip a finger on the surface of a tissue consisting of five different layers in such a way that all of them are invaginated beneath into a test tube the tip of the finger will necessarily carry at its most advanced part the layer which is most superficial, which in the case of the surface skin consists of the keratinized horny or scaly layer of the epidermis. In the course of time this tip may get separated from the main body of the test tube, the scaly end with all the structures being isolated as an island; a section through which will necessarily carry the keratinized cells whorled in the middle or at the centre, surrounded by the successive layers of the epidermis from the centre outwards exactly in the same order as they are found in a normal epidermis from the surface beneath. Thus in a section of a cancer they appear in three distinct layers as described above.

Sometimes cell-nests are exceptionally seen in tissues other than squamous-celled carcinoma; *e.g.*, in the normal **tonsils**, and **buccal cavity**. This exception is of considerable diagnostic importance to avoid mistakes in diagnosis.

The characteristic **clinical types** of Squamous-celled Carcinoma met with are the following:—

Clinical types.

(a) **Malignant Papilloma**, or **Malignant papillomatous Wart**. In this type the warty growth is more superficial than deep, the destructive process is limited, the growth appears like a projecting cauliflower resembling somewhat like a non-malignant papilloma, which may have been pre-existing. The individual masses are soft and slippery and bleed very easily on even a simple touch. The peculiarity of this type is little destruction but too

(a) Malignant papillomatous type.

much surface proliferation. *Vide* photo-plate No. XLIV malignant papillomata on the lips.

(b) Nodular type.

(b) **Nodular and Indurated Form.**—This constitutes of a mass with central crateriform ulceration. This form gives rise at first to an elevated mass, the surface of which instead of being papillomatous appears like mole-hills which are nodulated and situate all round the outer half of the growth; the inner central area is depressed. Ulceration occurs by central degeneration with foul smelling discharge giving rise ultimately to a crater. The edges of the ulcer are hard and everted. This destruction in the central zone proceeds more slowly than the new-formation at the outer zone. *Vide* photo-plate No. XLV.

(c) Ulcerative forms.

(c) **Ulcerative Form.**—This type remains confined more to the surface of the skin than any proliferation above or beneath it; and as such closely resembles a Rodent ulcer. The destructive process extends equally with the new-formation giving an appearance of a depressed sore with sharply cut edges. *Vide* photo-plate No. XLII of an engine driver, and photo-plate No. XXXIII carcinoma of the skin arising from a keloid.

(d) Depressed and indurated base with little surface ulceration. Lip-cancer type.

(d) Lip-cancer type with **depressed base** and **little** ulceration. This type does not proliferate above or on the level of the surface, but extends underneath the skin like a fir tree growing in an inverted way. The flattened roots proliferate on the surface but the branching excrescences burrow and extend underneath. The fibrous **stroma** contracts and compresses the columns of epithelial cells, making the surface wart-like and indurated with little-ulceration but with pronounced **depression**. The base therefore becomes very hard. The progress of proliferation and new-formation of the tumour cells underneath the surface is less rapid than in other forms. This is more common on the lip, *vide* photo-plate No. XLVI, twin brother of case illustrated in photo-plate No. XLIV. None of the brothers saw an English smoking pipe in



DEPRESSED CANCER OF LIPS.



their life time. This type disseminates extensively by permeation of the lymphatics, and soon infects the neighbouring glands.

In all the above clinical types of **squamous-celled carcinomata**, the pathology and clinical symptoms are similar.

All the clinical types are pathologically similar.

**Clinically**, the squamous-celled carcinoma are met with, as described above, on the skin, buccal cavity, tongue, pharynx, larynx, œsophagus, anus, pelvis of the kidney, ureter, bladder, the vagina, the vulva and cervix uteri. Carcinomata of other types to be described below are sometimes transformed into squamous-celled variety. When transformations into malignant conditions of other neoplasms, chronic ulcers, or granulomata, occur they usually take to squamous-celled carcinoma in form.

Clinical features.

Sites.

Malignant transformations.

The nature of the onset is insignificant, unless there is a pre-existing chronic ulcer from long-standing irritations of some kind. It starts from a point exhibiting a nodular or papillomatous or warty nature and then generally extend; the most central part breaking down and ulcerating by degenerations. The neighbouring lymphatics are soon affected and the glands become the seat of exactly similar growths later on, but not in the early stages when they are merely matted. The glands thus affected are fixed in the surrounding tissues, and if superficial, **ulceration** soon follows by the involvement of the skin. With the further progress of the disease, more distant groups of lymphatic glands are invaded; but **dissemination** of the squamous type in the substance of the **internal viscera** is **unusual**.

Starts in a point.

Extends

Lymphatic infiltration.

Ulceration.

Internal dissemination unusual.

**Degenerations** of cystic nature sometimes occur especially in the neck; and if operated on mistaken diagnosis a thin turbid fluid or discharge of sero-purulent nature escapes; which may be sometimes mixed with white masses of epithelial debris; ultimately resulting into

Degenerations cystic.

Extension to main vessels. Fatal hæmorrhage. Cachexia. Exhaustion death. fatal sinuses. Such extensions and ulceration may implicate the main vessel of the neck and at any moment terminate the scene by fatal hæmorrhage. In any case death occurs by exhaustion and cachexia as a result from the absorption of sepsis and toxin.

Treatment. **Treatment.**—Local treatment varies according to site, for the details of which the reader is referred to regional surgery. For general treatment *vide* below.

For the convenience of description one other form of blastoma, *viz.*, the Rodent ulcer is described here, as it is very similar in nature to squamous-celled carcinoma.

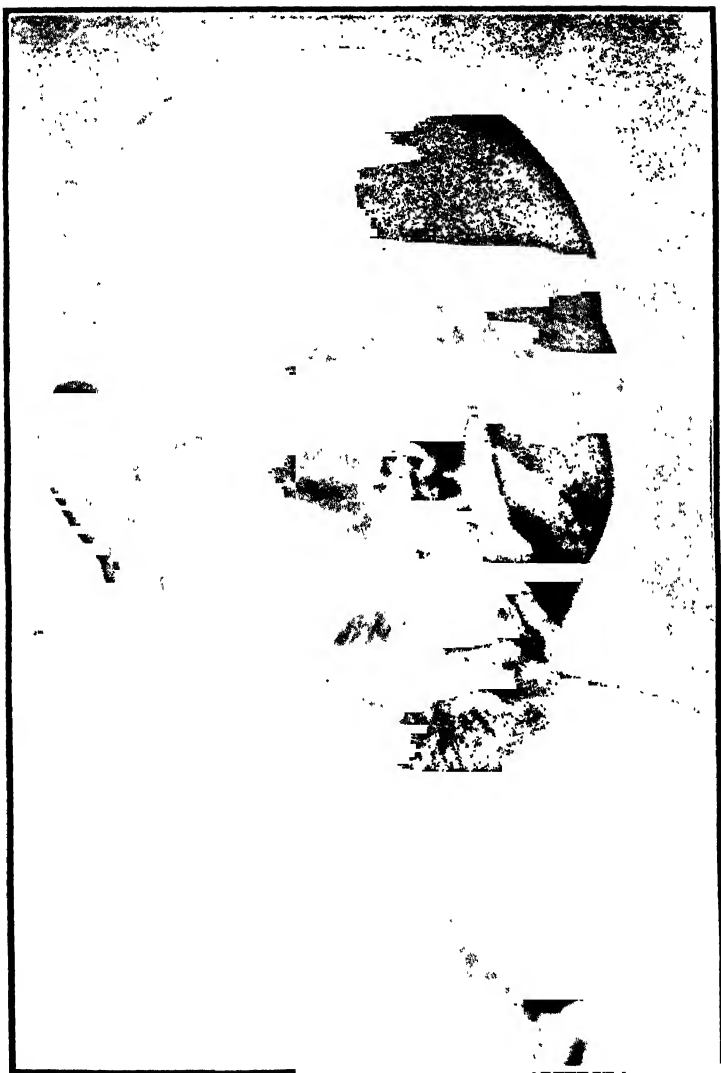
## 2. Rodent Ulcer.

## 2. RODENT ULCER.

**Rodent Ulcer.**—Also wrongly described as Rodent cancer. Rodent ulcer is a chronic form of carcinomatous condition on the skin. It starts from the basal cells of the epidermis. It is usually seen on the face and most commonly at the inner canthus of the eyes on the side of the nose (*vide* photo-plate No. XLVII). It is also seen at the outer angle of the eyes, forehead, lips, chin and on the malar bone. It rarely grows on the neck and trunk, but it is never seen on the limbs.

**Situations.** The exact tissue of its origin and first appearance is still a disputed question. It is generally believed in England to start from the hair follicles; sebaceous and the sweat glands are also supposed to be the tissues affected. In the Continent it is believed that the ulcer grows from the basal cells of the epidermis, and is therefore described as Basal-celled carcinoma. Gradually in the course of time it affects the dermis. The epithelial cells appear like large irregular flask shaped ingrowths separated from each other by many normal papillæ, but the solid columns all reach to about the same level. The surface epithelium does not proliferate. The dermis and subcutaneous tissues are infiltrated with the columns of the irregular growths of small, round and oval cells. The rodent cells are far smaller than ordinary epitheliomatous cells, and

**Morbid histology.**



RODENT ULCER.



are compact cells with scanty protoplasm having a darkly stained nucleus which is very different in appearance from the *pale* and *inflated* cells of epithelioma. They do not become horny or exhibit any sign of keratinoid changes; they tend to vacuolate and form spaces in the ingrowing mass. There are:—

- (i) No “cell-nests.”
- (ii) No prickle cells.
- (iii) No keratinization.
- (iv) No metastasis.
- (v) No gland involvement.
- (vi) No round-celled infiltration.

The epidermis being infiltrated is soon destroyed, which allows the cancer cells to protrude out by disintegration and ulceration. A typical squamous carcinoma is essentially an ingrowth from the epidermis into the dermis. A rodent cancer starts from the epidermis and then being fully developed there, disintegrates and ulcerates out on the surface from below. The external layer of cells therefore remains distinctly columnar in shape.

Age of **onset**.—Generally it develops after thirty-five or forty years of age, but cases are seen started as early as at sixteen. Rodent ulcer is as common in India as it is in Europe. Onset.

It starts as a small pimple covered by shiny smooth and thinned epidermis, which soon ulcerates. The ulceration progresses as rapidly as each of the pimple ulcerates, giving no time to form into a large mass of growth. At some part of the edges sometimes it shows signs of temporary healing covered by an unstable scar which soon again breaks down. Clinical features.

It is a growth of chronic character and the progress is very slow. Cases usually give history of suffering for fifteen, sixteen or even twenty years with partial destruction of eye balls or nose. A typical ulcer is a flattened sore; with the **base** exhibiting no sign of healthy

granulation, but is smooth and shiny. The **discharge** is thin and purulent. The **edges** are irregular and consist of well-defined rolled border which constitutes the peripheral portion of the ulcer which had not yet broken down. There is practically no pain complained of in rodent ulcer but it is itchy. It gradually extends and destroys all surrounding and deeper tissues such as the bones, the eye lids, the eye ball sometimes resulting in hideous deformities.

Secondary growths in the lymphatic glands never develop. Death occurs from meningitis, erysipelas, hæmorrhage, and septic complications of the lungs, etc., or from senility of old age. Dissemination of the Rodent ulcer to other viscera is not known, and absence of "cell-nests" is its characteristic distinguishing feature from skin cancer.

**Differential diagnosis.**

**Differential diagnosis.**—Rodent ulcer is mostly mistaken for Lupus, Syphilitic ulcer, and Squamous Epithelioma. Lupus is a disease of earlier age. The situation, the apple jelly nodules, and the absence of rolled margin, the giant-cells, the presence of tubercle bacillus, are characteristic features to distinguish Lupus from Rodent ulcer. Microscopical examination should always be made to confirm it. Syphilitic lesions are characterized by other local and constitutional syphilitic signs, Wassermann's reaction in the blood, more rapid growth, and lack of characteristic margins. Squamous-celled Epithelioma invades the glands and are more fatally rapid in growth.

**Treatment.**

**Treatment.**—The soonest the ulcer is completely excised out the best, and after an early operation cure is also sure provided the size is small. Next to excision the best method is radium therapy. This is the only kind of so called cancer where complete cure has been claimed by many surgeons. Application of X-rays is also successful and least disfigurement is produced, although the progress of cure is very slow, and it always requires many exposures. Carbon dioxide snow has been used by skin

specialists with successful results. Diathermic cautery has been lately found to be of great value.

## B. GLAND TYPES.

### CARCINOMATA OF GLANDULAR EPITHELIUM. B Gland types :—

It must be understood at the very start that although the carcinomata of this group are called carcinomata of glandular epithelium many tumours of this class may not show any tendency to constitute themselves into a corporate body of a gland. And therefore this type may be histologically sub-divided into two groups. *Viz.* :—

(a) **Carcinoma Simplex**, that is those in which the constituent cells remain beyond showing any tendency to co-operate each other and arrange to form into a structure like a gland, but compose themselves into groups of solid alveoli or columns. These are generally composed of **spheroidal** or polygonal cells. The term *simplex* is used with reference to the *construction* and structure; being destitute of actual glandular arrangement, although constructed of glandular epithelium, or *materials* such as the spheroidal type of cells. (a) Carcinoma. simplex.

(b) **Adeno-carcinoma**.—That is those which exhibit some tendency to copy a gland structure, and their cells arrange to corporate into tubules or acini in such a manner as to recall the structure of the parent gland from which they arise. They are composed mainly of **columnar** cells; or **cubical** cells. These two kinds of epithelium change their form according to their capacity to attempt to functionate in secreting the material normally produced by the parent gland. The more columnar they are the nearer they are to normally secreting columnar epithelium. We have already explained how in normal thyreoid or breast the cubical epithelium becomes columnar during the period of secretion and then again revert to cubical when involution takes place. *Vide* n. 173. (b) Adeno carcinoma.

It must be remembered that whether the cancer develops into **simplex** type, or **glandular** type, the cells constituting them are of epithelium which are found in the **glands**, and therefore carcinomata exhibiting such cells are often described as the **malignant** form of **adenoma**, or a carcinoma whose structure very closely resembles that of a normal gland. By that it should not be understood that all spheroidal-celled or columnar-celled carcinoma are malignant transformation of glandular type of non-malignant tumour known as Adenoma; or even a tumour arising from a secreting glandular tissue of a normal parent gland.

Pathological types.

The different forms of this type of Carcinoma are the following :—

(i) Spheroidal celled carcinoma. These are malignant forms of adenoma simplex type.

(i) **Spheroidal-celled** carcinoma.—This type grows from the glandular epithelium of organs composed mostly of secreting glands, *e.g.*, thyroid, breast, prostate, pancreas, salivary glands, kidney, testicle, ovary, and stomach. The shape of the new cells of the carcinoma differs according to degree of malignancy, but it is mostly like the typical secreting epithelial cells of a parent secreting gland, *e.g.*, they are spheroidal, or cubical, or polygonal. The glandular *structure* reproduced by them, is atypical, and of **simplex** type; but gradual or abrupt transition may take place to such an extent that they may be described as **adeno-carcinoma**. The departure of these cells from the normal cells lies in its failure to produce *secretion*, or in the **absence of proper secretion**, and in the *alveoli* formed of these new structures; the cells making them being **atypical**, are **irregular in shape** and **arrangements**. The more malignant a growth is the more indifferent are its cells. The glandular acini from which a carcinoma originates are **not** supported by any **basement membrane**, but the cells encroach upon surrounding parts by travelling beyond it along the lymphatics, transforming all tissues by **inclusion** into tumour substance. Pathologically we differentiate the various types of carcinoma of glandular epithelium according to the morphology of

their cells; but clinically it is customary to classify them with respect to certain gross characteristics according to their apparent clinical conditions produced by: either, (a) abundance of **fibrous stroma**; or (b) abundance of **cellular parenchyma**; or (c) presence of **colloid degenerations**, of mucoid nature, etc., in the substance of the growth.

**The clinical forms of Carcinoma Simplex** met with are the following:—

Clinical forms of Carcinoma Simplex are:

### (1) THE SCIRRHOUS TYPE.

(1) The Scirrhus Type.

In this type due to its **chronic**, long extended, and **slow** course of formation of the neoplasm the fibrous **stroma** develops in abundance which may avail of the opportunity of abundant supply locally, as in the breast. Such excessive proliferation of the fibrous tissues makes the growth **hard** in consistence, and is therefore described as "Scirrhus." It should not be understood that a Scirrhus Carcinoma is always a Carcinoma Simplex, and a superabundance of stroma may take place in glandular adeno-carcinoma and squamous-celled carcinoma also.

Scirrhus Carcinoma is met with in the **breast** in the majority of the instances, but it is also found in prostate, pancreas, and pylorus. On naked-eye examination a scirrhus cancer appears as a nodular, hard, and a tough mass, with an imperfect limitation at the surrounding neighbourhood. On section it creaks under the knife as one feels when cutting an Indian pear,—“Naspati.” The cut surface becomes concave owing to the contraction of the fibres of the stroma, which in colour appears yellowish-white. On scraping the surface with the blade of a scalpel a typical cancer juice with floating oily globules may be obtained. Under microscope this juice exhibits typical cancer cells and debris.

Microscopically, in a mammary Scirrhus, alveoli of all shapes and sizes are found. They have no regular arrangement, and they are packed with a variable number

of cells of spheroidal or polygonal shape, which have well-defined large deeply staining nuclei, and a moderate amount of cytoplasm; but **no intercellular** substance could be made out. Between the alveoli the fibrous tissue develops in a variable degree at different areas; *e.g.*, it is plentiful and dense in the central older portions of the growth, but more cellular, more scanty, and more delicate at the younger portions in the periphery. The more abundant the fibrous tissue the harder becomes the tumour, and slower it is in growth; and more consolidated the mass becomes in its formation. The growth extends in all directions along the lymphatics, and a round-celled infiltration of the surrounding tissues is always evident.

Degenerations.  
(a) Cystic.

**Degenerations** in the centre sometimes change the consistence of the tumour considerably, *e.g.*, (a) fatty degeneration, is often present which may produce small **cysts**. (b) Where the stroma becomes very excessive the cell elements may be deprived of its nutrition caused by the compression of the nutrient vessels; and thus producing **atrophic** degenerations, when it is described as a special clinical variety called **atrophic scirrhus**.

(b) Atrophic Scirrhus.

(2) The Encephaloid.

## (2) ENCEPHALOID, MEDULLARY, or ACUTE CANCER.

In this type due to the **acute** hasty invading nature of the growth, the neoplasm finds little time or favourable conditions to consolidate firmly by the formation of fibrous stroma, but is supplied by very vascular canalizations with blood. The **cellular parenchyma** hurriedly multiplies and develops in abundance. Such excessive proliferation of the cells with so abundant hæmorrhagic supply makes the growth **soft** resembling something like the brain substance in consistence, and is therefore clinically described by the term "**medullary**," or "**encephaloid**."

Site.

The Medullary or Encephaloid carcinoma is met with in the **testis**, **breast**, kidney, and a few other





TESTICULAR TERATOMA.

glandular organs. On naked eye examination the surface of a section exhibits a soft whitish mass, somewhat resembling brain substance as stated above. It is very vascular and sometimes pulsating. Extravasation of blood in some areas inside the tumour substance is not unusual.

Clinically it is manifested by a rapidly growing tumour, softer in consistence than the Scirrhus, and extensively adherent to the surrounding tissues. It is freely supplied with blood channels. The tumour cells are disseminated very early into the neighbouring lymphatic glands, and thus infiltrate into the surrounding muscular, vascular and other tissues. The surface of the overlying skin becomes stretched and shining, with dilated blue veins apparently visible and prominently displayed on it. Ulceration soon occurs which exhibits a  
 a characteristic foul surface, known as **fungus hæmatodes**,  
 clinically manifested by a foul smelling, bleeding, fungating growths, sprouting luxuriantly like big masses of proud flesh. *Ulcera-  
 Fungus  
 Hæma-  
 todes.*  
*vide* photo-plate No. XLVIII.

### (3) COLLOID CANCER.

(3) Colloid  
Cancer.

By Colloid Cancer we should not conceive the idea that there is any particular pathological type of cancer. It is a *degeneration*.

In some cases of Spheroidal-celled carcinoma mucoid degenerations of extensive **colloid** nature occurs. These are especially expressed clinically as **colloid cancer**. Colloid degeneration is also met with in other types of glandular or Columnar Carcinoma described below and therefore it cannot be claimed to be present in tumours of any particular type of epithelium. In this clinical type the alveoli become more or less filled with mucoid material of colloid nature, which may not exhibit a similar degree of degeneration in every part of the tumour.

Site.

Colloid Cancer is met with in the breast, and digestive tract; more frequently arising from the stomach, intestine, or omentum.

Naked-eye examination presents an alveolar structure having the spaces filled with translucent gelatinous mucoid masses of varying density.

Microscopical examination shows little or no evidence of the epithelial cells which are rarely distinguishable; being replaced by structureless mucoid substance, excepting in the growing margin where the degenerative process is not yet completed, which helps the cells to be spotted. Some of the cells which are in the process of degeneration may exhibit their nuclei pressed on one side of the cell protoplasm by the globules of the degenerative materials just forming inside, in the body of the cytoplasm.

Columnar-celled  
Carcinoma.

## (ii) COLUMNAR-CELLED CARCINOMA or ADENO-CARCINOMA.

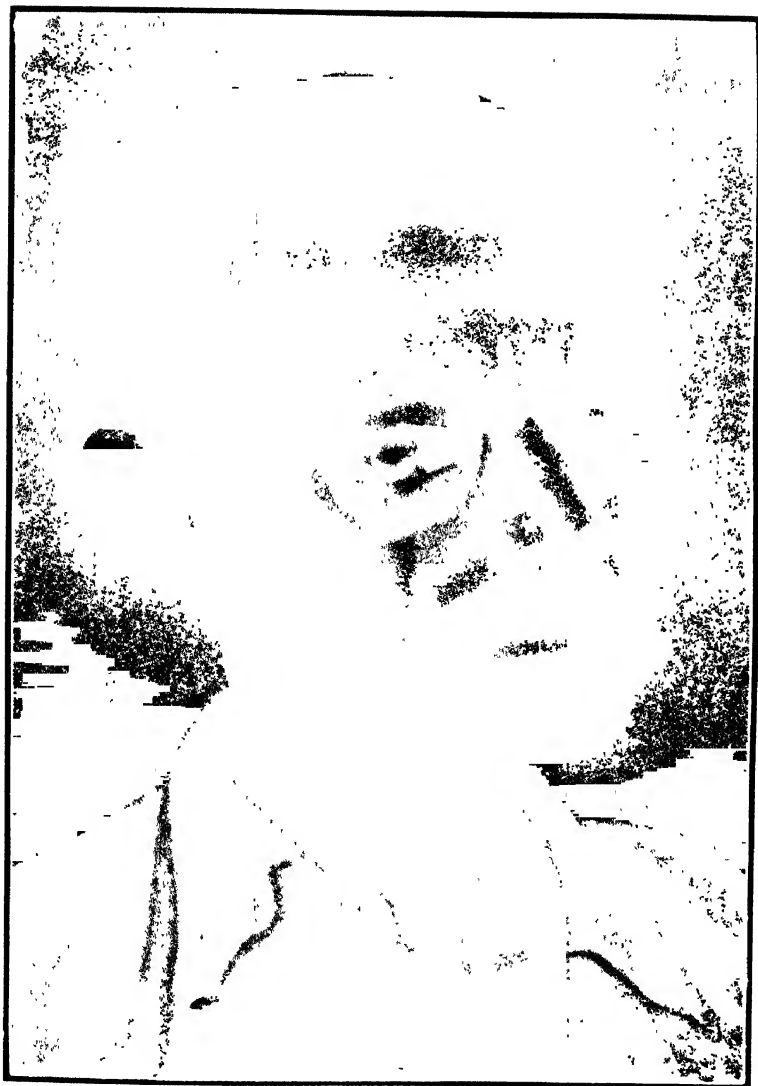
In this variety of carcinoma the epithelium constituting them is of **columnar** kind, and these cancers grow wherever the epithelium normally covering the tissues is of columnar variety such as in the **Lieberkuhn's follicles** or in the **ducts** of glands.

Columnar carcinoma is met with in the digestive tract, from the **pylorus** to the **rectum**. It is also less frequently seen in the stomach. The **ducts** which are the seats of these cancers are the ducts of acinous glands, such as that of the **mamma** (which is especially described clinically without any justification to be particularly called as **duct cancer**), the bile-ducts, uterus, and occasionally in the respiratory tract, and tubes of the tarsal (Meibomian) glands. *Vide* photo-plate No. XLIX.

Duct  
cancer.

This is  
the true  
type of  
glandular  
carcinoma.

This form of tumour in the majority of cases is a true **glandular** cancer, mimicing a secretion of atypical nature. The neoplasm may grow into a large size, and



CANCER OF MEBOMIAN GLANDS.



in the intestine the growth usually involves the whole circumference obstructing the lumen by stenosis.

The neoplasm forms a projecting growth from the level of the surface, and also penetrates deeply into the submucous and muscular coat, and is usually an overgrowth of **Lieberkuhn's** glands. This latter phase of its invasion characterizes its malignant nature and differentiates it from non-malignant columnar-celled adenoma, *vide* chapter III of this volume. P. 166.

Microscopically, the neoplasm consists of numbers of **tubes** and alveoli. The deeper portions of these neoplastic processes retain an imperfect alveolar arrangement, and between them a certain amount of **stroma** is observed. Although the individual parts of the growth adopts a **tube-like** nature of construction, it is irregular in size, irregular in shape, irregular in copying any original nature of the secreting epithelium from which the neoplastic proliferation commences. It is lined with **only** a **single** layer of **columnar-celled epithelium**, which manifests a regular arrangement or in places showing a tendency to proliferate. It must be distinctly noted that except for the fact that the deeper processes of the alveoli invade and destroy the deeper muscular layers and are irregular in shape, there is **no** other means of **distinguishing** a Columnar-celled **carcinoma** from a simple **adenoma**, or as a matter of fact from a simple **hypertrophy** or **hyperplasia**. A simple adenoma of the Lieberkuhn's gland may be easily distinguished from an adeno-carcinoma by the former's having completely and regularly lined alveoli with a layer of columnar epithelium, whereas in adeno-carcinoma the alveoli vary in shape and the epithelium is less regularly arranged, and above all, the main difference is manifested by the extension of the glandular tissue **into and between** the muscular fasciculi. The confirmation of this feature is possible if the whole thickness of the gut wall is available for examination.

Sometimes very difficult to distinguish a carcinoma from an Adenoma or a Hypertrophy.

The **stroma** consists of a delicate connective tissue. In a chronic case this stroma is abundant and **fibro-**

**cicatricial** in quality, but in the rapidly growing and softer forms the stroma is scanty in amount and **cellular** in nature. In some instances the projections of these epithelial walls into the cavity of the alveoli in the form of processes, might give them an appearance of **papillomatous** or **villous** carcinoma; or might produce a **polypoidal sessile** or proliferating **cyst-adenoma** by dilatations and distensions of the lumen, allowing the epithelial columns to project into the cavity along with the proliferation of the **stroma**, or ending in a sloughing **ulcer**.

In the **duct cancer** of the **breast** proliferations of the nature of cyst-adenoma, and formation of small cysts containing secondary intra-cystic papillomata are seen in a minor degree.

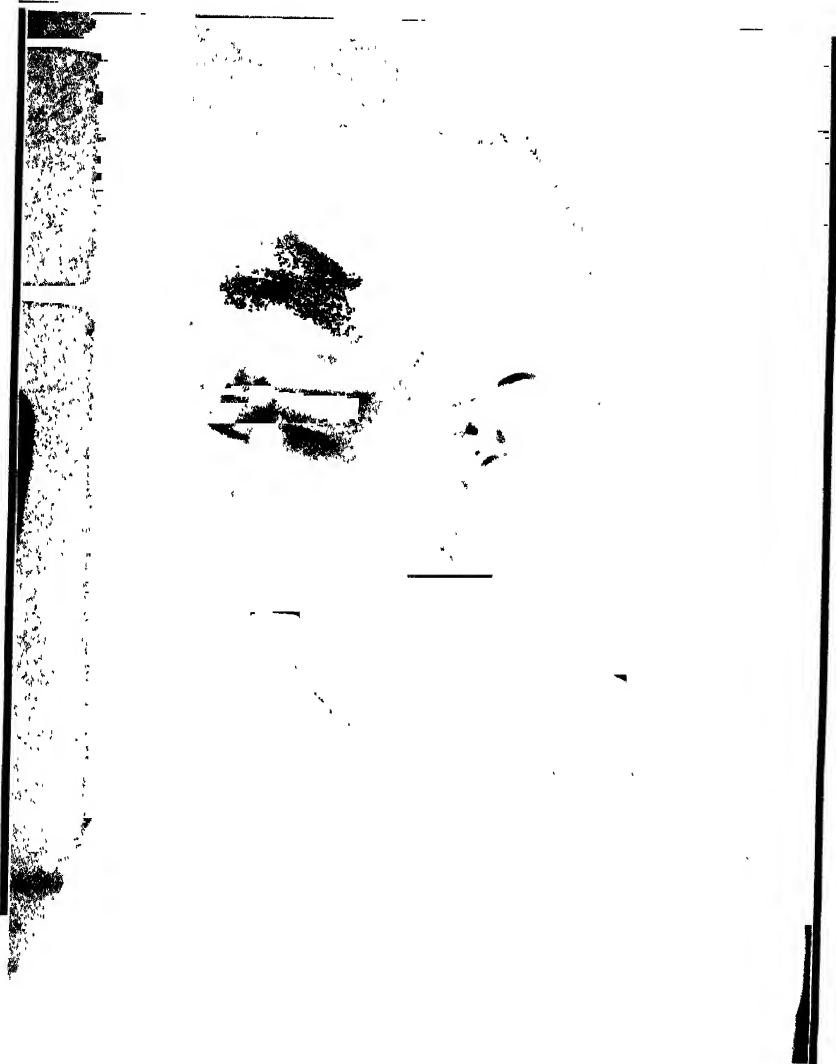
**Ulceration.** **Ulceration** often occurs giving rise to typical malignant ulcer characterized by indurated and everted edges.

**Extension.** **Extension.** The lymphatic glands at the neighbourhood are soon implicated. Dissemination of secondary colonies takes place at various viscera and in the same organ. Neoplasms of similar type of growth may occur in the breast, cervical portion of the uterus, and liver.

Columnar-celled Carcinoma is also met with in the maxillary bone. This may originate from the tubular glands of the mucous membrane which lines the antrum. *vide* photo-plate No. L.

**Degeneration.** **Degeneration.** Colloid degeneration of the columnar cells may occur as in the case of Spheroidal celled carcinoma already described.

**Keratinization.** **KERATINIZATION.**—Keratin is a substance which is like beeswax. It belongs to sclero-protein (protein of skeletal origin of insoluble nature) group. It is found in the surface squamous layers of the epidermis, in hairs, horns, hoofs, and nails. It is a horny material, very insoluble, and contains a high percentage of sulphur.



CANCER OF MAXILLARY GLAND.



Keratin is found in the stratum corneum formed from keratohyalin derived from the stratum lucidum, the latter obtaining from eleidin produced in the protoplasm of the cells of stratum granulosum. The continual shedding of the scales of the skin is in reality a process of excretion of sulphur removed from the body by transformation of the protoplasm of the cells of the deeper layers of the skin, the protoplasmic mass being transformed into keratin.

Where keratin is formed?

Keratin formation is a process of excretion of sulphur.

Keratinization increases in proportion of friction or irritation. In the tongue for instance drinking of strong spirits, constant smoking of irritating hot pipe, predisposed by syphilitic conditions, may excite proliferation and heaping up of the cornfield or keratinized epithelium. Or in other words the more the friction and irritation the more the keratinization occurs in the squamous cells, and the more is the leukoplakik degeneration. We must note that this is expected to happen where we have got squamous epithelium normally, *c.g.* :—

Keratinization may degenerate into Leukoplakia.

The more the friction and irritation the more is the leukoplakik degeneration of Keratinization.

(i) The skin.

(ii) From the mouth to the lower end of the oesophagus, including the whole of the pharynx, but *not* further down.

(iii) Vagina and cervix uterus.

(iv) Pelvis of the kidney, ureter, and bladder. In the latter situation it is transitional stratified.

In the tongue and bladder this leukoplakik keratinization is often seen, which is clinically described as Leukoplakia of the tongue, bladder, etc.

This condition may form the starting point of epithelioma or squamous carcinoma.

Excepting these positions squamous cells with keratinization and "cell-nests," seen any where else in any other organ and tissue must atone be concluded to be due to migrated metastasis, secondary to squamous carcinoma elsewhere.

Mixed  
Squamous-  
celled and  
glandular  
carcinoma.

**MIXED SQUAMOUS-CELLED AND GLANDULAR CARCINOMA.**—On the glandular or columnar-celled epithelial surfaces on the other hand, we do not expect to find keratinization because there is no squamous layer over it. But if a constant friction and strain pass over the columnar-celled layer of epithelium, the irritation may excite **metaplastic potentialities** of epithelial cells and a change may occur from columnar type of cells to squamous type, which would manifest keratinization at extremely unusual sites, *e.g.* :—

Metaplastic  
Potentiality.

#### METAPLASTIC POTENTIALITY.

(i) In the gall-bladder, a mixed type of carcinoma known as **mixed squamous-celled and glandular** carcinomata is seen as the effect of metaplastic change from columnar to squamous type.

(ii) In the urinary bladder also, when leukoplakia occurs, by metaplastic change from transitional to squamous type, it may form the starting point of squamous-celled carcinoma, composed of islands of cells of the transitional or stratified type; but prickle cells are absent and there is rarely any pronounced type of horny degeneration.

(iii) A form of squamous carcinoma also occurs in the antrum of Highmore, the cells of which may be extremely *atypical*, *vide* photo-plate No. XLIII.

We should always remember that keratinization or horny degeneration is dangerous, as it may :—

- (a) Excite Leukoplakia;
- (b) Followed by squamous carcinoma, or;
- (c) Excite metaplastic potentialities and produce mixed squamous celled and glandular carcinoma at places where there is no squamous layer of cells normally present.

**CLINICAL FEATURES OF CARCINOMATA.**

Clinical  
Features of  
Carcino-  
mata.

There is a well recognized group of signs and symptoms which when it can be established, in whole or in part, is generally indicative and pathognomonic of the development of malignant growths. These are :—

(1) A definite **history** of a **strike** or **trauma** or some irritation which can be secured if especially sought for in the majority of the cases.

(2) The presence of a **tumour**, if it develops at the accessible areas of the body, which is **painless** at the initial condition.

(3) **Rapid Growth** of progressive nature not yielding to any treatment.

(4) **Anæmia** and emaciation.

(5) Age.

(6) Pain at the **seat** of growth or referred.

(7) Loss of **weight** and cachexia.

(8) Disturbance of **function** of the particular organ or tissue affected.

(9) **Hæmorrhage**.

(10) Sero-Sanguinous **discharge** when cutaneous or mucous surfaces are affected. **Ulceration** of fungoid nature.

(11) Enlarged lymphatic **glands**.

(12) **Adhesions** with surrounding structures

**DIAGNOSIS.**—For diagnosis *vide* last chapter. Diagnosis.

**TREATMENT OF CANCER.**—It is a common experience of all experienced surgeons that an early thorough operation effects a cure in many cases. In this respect the public require a thorough education. The chances of cure in early cases and the danger of delay should be made to be realized by the public very thoroughly.

Treatment  
of Cancer.

For clinical purposes the method of treatment depends on two factors, *viz.*—(a) whether the tumour is accessible, or inaccessible; (b) according to the advancement and progress of the disease and in what stage and condition the case is first seen. Generally the cases may be grouped under the following classes, *viz.*:—

- (i) Clearly operable.
- (ii) Doubtfully operable.
- (iii) Radical operation impossible.
- (iv) Hopeless.

1. Operation.  
In clearly operable cases.  
(i) Excision.

### (1) Operation.

In accessible and early clearly operable cases, the treatment consists in its removal 'en bloc,' as *early* and as *completely* as possible. The excision must include a good margin of apparently normal tissue, for the details to be followed in individual cases, *vide* regional surgery. Skin, glands, the whole area of lymphatic permeation and all other tissues which are suspected to be complicated with the growth, must be removed *thoroughly*.

(ii) Amputation.

In the cases where a limb is so affected that it is doubtfully operable high amputation should be the course adopted.

Partial operation in doubtfully operable cases, helped by radio-therapy.

In the cases where the tumour is not wholly accessible, or where due to extension complete removal is impossible, it is wise to remove the accessible portion and control the hæmorrhage by the use of diathermic knife, cautery and styptics; helped by other procedures of treatment such as radium therapy, as a supplemental measure to destroy the diseased cells which escaped the surgeon's knife.

The other procedures helpful to treat cases, especially the inoperable and hopeless cases, are:—

2. Radium Treatment.

(2) **Radium Treatment.**—The radium may be buried in the body of the tumour for twenty-four hours or more according to the amount of radium used, and the size and consistence of the tumour. In the superficial carcinomata and sarcomata radium

has its highest application and effectiveness. It should be noted that different types of growths are differently sensitive to radio-activity, and radium has its own limitations. *Vide* Minor Surgery.

(3) **X-Rays** are indicated in the same nature of 3. X-rays. cases as Radium. For the details, *vide* Minor Surgery.

(4) **Diathermy** differs radically from Radium and X-rays therapy; and it is more and more largely used in the treatment of cavities. Its purpose is the direct destruction of the diseased cells rather than their physical or chemical modification. It is in fact a supplementary surgery. It differs from other methods of thermotherapy, in that it does not transmit heat, but generates it in the tissue itself using the tissue as the factor of resistance. **Malignant cells are more vulnerable to heat** than normal cells, and therefore with a lower heat than that which coagulates normal cells the migrating tumour cells can be destroyed. The rapid coagulation of the malignant cells helps in arresting metastases. Recently Diathermy and Radium therapy are securing more favour than other forms of treatment as these methods are gaining more confidence of the surgeons. For details of Diathermy, *vide* Minor Surgery.

Injection of **Coley's Fluid**.—Coley's Fluid had been tried and successful results have been recorded by many well-known surgeons. The initial doses should be very small as the reactions are alarming and the Fluid is very toxic. The injection should be commenced from a dose of half a minim or less, increasing it to 7 or 8 minims in **different weekly courses** of treatment prolonging to several weeks. The fluid consists of, as described in volume I, sterilized culture or streptococcus pyogenes and micrococcus prodigiosus in bouillon. The experience of the majority of the surgeons with this fluid had not been encouraging. But many older authorities advocate its use in Sarcomata. Retrogression in the Sarcomata is sometimes observed after

Coley's fluid.

an attack of erysipelas; and this treatment is based on that observation.

All the above forms of treatment have limitations and in this connection the following measures taken in time may be of value; *viz.* :—

(1) Precancerous lesions, such as warts, moles, *nævi*, keratoses, and slowly healing fissures, should be destroyed by electro-coagulation (diathermy); and in this way cancers could be prevented in many cases which may have a chance to develop later on.

(2) Every case of epithelioma should be thoroughly treated and completely eliminated 'en bloc' at once. It is unwise to attempt to destroy a cancer piecemeal.

(3) Radium, properly applied, is the best single agent in the treatment of all forms of cancer.

(4) In all well-advanced cancers two or more methods, *e.g.*, operation and radium should generally be combined to advantage in the complete elimination of the disease.

(5) Skill in surgery and pathology, keen judgment, thorough knowledge in anatomy, and careful consideration of the individual patient, count for much in this field, as they do in every branch of medicine.

General  
Treatment.

"Whether as a cause or as result of the disease, every patient suffers from a general systemic debility which becomes part and parcel of the disease, helping to form a vicious circle in its course, and, therefore, demands medical attention. The whole patient is ill with cancer. The anæmia, the cachexia, the emaciation, all point to serious debasement of the blood, and other body tissues. Whether this be regarded as cause or consequence, and whatever surgical or semi-surgical measures be employed, it is evident that the patient will stand a greater chance of reacting to treatment and overcoming the disease if his system be furnished with needed nutrient elements, body salts, etc., his metabolism improved, and all of his vital functions judiciously stimulated."

We have already pointed out the defect of calcium salts observed in all cancerous conditions. For this purpose hypophosphites of lime in the shape of some organic preparation is of great value. Dietary measures, attention to body habits, tonics, etc., are factors the necessity for attention to which is self-apparent.

There is no disease where we could afford to neglect the general and constitutional treatment, and this medical phase vitally concerns itself with the treatment of the patient's general condition.

### ODONTOMATA.

Teeth are derived from epiblastic cells, and in connection with the teeth many kinds of neoplasms are observed. Some of them are proved recently to be blastomatoids, some are benign in nature, but many are distinctly malignant. Bland-Sutton has classified the tumours in connection with the teeth or teeth-germs under seven distinct types. But all the varieties described by him are not seen in the human. Those tumours originating from some pathological conditions of the teeth or teeth-germs are clinically called **odontomes**.

The commonly known important varieties of Odontomes including both non-malignant and malignant forms met with in man are the following:—

#### (1) **ADAMANTINOMA** or **EPITHELIAL ODONTOME**. (1) Adamantinoma.

This is a rare type of Odontome in which a peculiar blastoma arises from the primitive enamel organ. This enamel is an epithelial structure. The tumour is at first non-malignant in nature but as it is surrounded by a thin capsule of shell-like bone which may eventually rupture, it may extend into the antrum and the orbit and turn locally destructive and malignant. Clinically in the majority of the instances it arises in the upper jaw and may be solid or cystic. As a rule it

remains small in size. Sometimes it attains a considerable size as large as a child's head.

Microscopically, it consists of broad branching columns of epithelial cells surrounded by a dense fibrous stroma having the outer layer of cells cylindrical or columnar in type, the inner ones becoming flattened. The centre of the mass gradually turns cystic by degeneration giving it a glandular appearance.

(2) Compound Follicular Odontomata.  
Tooth implicated as dentigerous neoplasms.

(2) **COMPOUND FOLLICULAR ODONTOMATA**, are an anomaly of development of **tooth**, and these neoplasms are formed of various tissues which enter into the formation of the tooth, growing and proliferating in the neighbourhood of the jaw. In a single tumour a large number sometimes as many as forty ill-formed tooth have been found (Choyce). They grow to a considerably large size and many show an association with, and exhibit the nature of the **osteoma of the antrum**.

### SUMMARY.

Malignant Neoplasms. Malignancy, its clinical and pathological meanings. Seven Signs of Malignancy.

Characteristic pathological features of malignancy:—

- (i) Formation.
- (ii) Progress of Growth.
- (iii) History of Incidence.
- (iv) Age.
- (v) Capsule.
- (vi) Vegetative or Embryonic character of cells.
- (vii) Mitotic figures.
- (viii) Infiltration.
- (ix) Metastatic growth.
- (x) Formation of blood vessels.
- (xi) Recurrence.

**DISSEMINATION** and Metastases.

**METHODS OF SPREAD.**

- 1. Infiltration.
- 2. Permeation.

Microscopic growing **edge**. Extent and space of permeation.

3. Transplantation.

4. Embolism.

The *degree* of Malignancy and their variation in secondary growths. The *consistence* of a malignant growth varies with malignancy. The *incidence* of Cancer. Age Incidence. Sex Incidence. Site Incidence. Education of the public. Warning of the Precancerous conditions. Buccal Mucosa, Uterus, and Cervix, and Breast, Penis, Skin, "Carcinosis" and "Pre-cancerous" condition.

(a) A general factor, and

(b) A local factor.

A. Preparation of a soil for the Growth.

Tissue Predilection in secondary growths.

### THE MALIGNANT ATYPICAL CYTOMATA.—

I. Epiblastic Cells.

II. Mesoblastic Cells.

III. Pigmented Tumours.

IV. Hypernephromata.

V. Teratomata and Organomata.

Essential characters of Carcinomatous growths. Encephaloid and Scirrhous. Development and terminations. Similarity with infective processes. Natural Immunity. Acquired Immunity. Natural cure. Clinical varieties. Clinical Types.

#### A. Surface types.

1. Squamous Celled Carcinoma.

Histology of the skin and their special features

(i) Layer of Keratin.

(ii) Layer of Kerato-hyalin.

(iii) Layer of Eleidin formation.

(iv) Layer of Prickle cells with Lymph Channels. Layer of Permeation and Pigments.

(v) Layer of Multiplication and Pigments.

Layer of Nævus cells. Melanin of Melanoma.

Cancer columns constituting the parenchyma and stroma. Cell-nests in the substance of the cancer columns. "*epithelial pearls*."

Clinical types.

(a) Malignant Papillomatous Type.

(b) Nodular Type.

(c) Ulcerative forms.

(d) Depressed and Indurated Base with little surface ulceration. Lip-cancer Type.

All the clinical types are pathologically similar.

2. Rodent Ulcer.

**B. Gland Types.**

Carcinoma Simplex.

Adeno-carcinoma.

Pathological Types:—

**(i) SPHEROIDAL-CELLED CARCINOMA.**

These are Malignant forms of Adenoma Simplex Type.

Clinical forms of Carcinoma Simplex are:

(1) The Scirrhus Type.

(a) Cystic.

(b) Atrophic Scirrhus.

(2) The Encephaloid.

(3) Colloid Cancer.

**(ii) COLUMNAR-CELLED CARCINOMA.**

Duct Cancer. This is the true type of Glandular Carcinoma. Keratinization. What is Keratin? Where Keratin is formed? Keratin formation is a process of excretion of sulphur. Keratinization may degenerate into Leukoplakia. The more the friction and irritation the more is the Leukoplakik degeneration of Keratinization. Mixed Squamous Celled and Glandular Carcinoma. Metaplastic potentiality. Clinical Features of Carcinomata. Diagnosis. Treatment of cancer.

Clinical Features of Carcinomata. Diagnosis. Treatment of cancer.

In clearly operable cases.

1. Operation.

(i) Excision.

(ii) Amputation.

Partial operation in doubtfully operable cases, helped by radiotherapy.

2. Radium Treatment.

3. X-rays.

Coley's Fluid. General Treatment.

**ODONTOMES.**

(1) Adamantinoma.

(2) Compound Follicular Odontomata. Tooth implicated as Dentigerous Neoplasms.

## CHAPTER V.

### SARCOMA MELANOMA HYPERNEPHROMA TERATOMA.

The term **Sarcoma** means flesh-like tumour. Sarcoma grows from the mesoblastic tissue and its cells resemble those which give rise to connective tissues in the embryo. At the very start it is interesting to note that inflammatory cicatricial new formations, or the **fibroblasts**, are also made of connective tissue cells of mesoblastic origin. These granulation tissue cells are also similar to embryonic type of cells which of course are ultimately transformed into mature tissues, such as fibrous, or osseous, etc. Both have the same kind of cells, immature blood vessels, and an ill-defined margin of growth. Both in the case of Sarcoma as well as in inflammatory granulation tissue the factor of exciting the proliferation of such cells may be traced from some kind of irritation in the shape of trauma, with the only difference that in the inflammatory granulation tissue no sooner the purpose is subserved the process of further growth which is responsive in nature, ceases, and even takes to retrogressive atrophy; whereas Sarcoma continues its process of growth indefinitely, although we are not aware whether such proliferation is responsive or not. Sarcoma of course may arise without any apparent cause, but the development of such a tumour after a trauma is a very common occurrence, or sequence.

A **neoplasm** of the type of **sarcoma** consists mainly of two principal parts, namely (i) a **parenchyma**; and (ii) **stroma**. The parenchyma is formed of atypical connective tissue cells which assumes the power of limitless multiplication. The stroma consists of fibrous tissue having three primary parts, such as: (a) **each** parenchyma cell having an envelop surrounding it individually, composed of a very delicate fibre like structure; (b) the

Two principal parts of the neoplasm called **sarcoma**, viz.:—  
(i) **Parenchyma** cells.

(ii) Stroma of fibrous tissue.

Stroma has three parts:—

(a) Delicate envelope around each individual cell.

(b) Net work inside the body supporting blood channels.

(c) Occasionally a capsule around the whole tumour.

Stroma.

main net work inside the tumour dividing it into **compartment** of parenchyma cells, in which in exceptional and rare instances there may exist blood vessels of perfect capillary type which are well formed, although generally these channels are mere interstices lined with endothelium; or simple blood spaces, only limited by the parenchymatous cells of the growth. Arteries and veins in fully formed condition are never found in these net works of the stroma. Lymphatics and nerves are absent. Thus metastases and dissemination take place mainly by blood vascular system in Sarcoma. To a certain extent it may disseminate by permeation *via* the lymph-channels, (c) occasionally a capsule around the **whole** tumour may, with difficulty, be made out.

It is according to the density, or otherwise, of this stroma that the Sarcoma becomes **hard** or **soft**. The more slow growing they are, the less malignant they are; and more definite is the formation of a simple capsule around them. The more rapidly growing they are, the more malignant they appear to be; and the less stroma they allow to develop; and thus the more malignant types become softer, vascular, and even pulsating. In some instances the soft vascular variety is very highly malignant and are clinically described as **medullary** Sarcoma. Dissemination is usually dependent upon the relation of veins to the tumour. As the blood interspaces in a Sarcoma directly communicate with the veins a malignant embolus very easily get access into the venous stream. We have described the further progress of such emboli previously. *Vide* Part II, Volume II. When metastases take place the secondary growths which may be very abundant, reproduce the structure of the original one.

Parenchyma cells.

The parenchyma cells exhibit various forms of different sizes and appearance. There is nothing peculiar in the character which differentiates the Sarcoma-cells from other forms of cells, and although as a rule mitotic

cell-division takes place, that is indirectly by karyokinesis, direct or amitotic division also occurs.

**CLASSIFICATION.**—It is often impossible to make out from which variety of connective tissue-cell the Sarcomata are derived. Sarcomata are essentially **cytomata** or cell-tumours, and the tumour cells practically never, or seldom combine to form distinct **tissues**; although in the more chronic or longstanding forms they **attempt** to do so, in which we find, in typical examples, the histological features are sufficiently characteristic, and the cells are sufficiently fully **differentiated** enabling us to adopt the more satisfactory **histogenetic** grouping corresponding to the non-malignant connective tissue tumours; there being no doubt that each typical connective tissue histioma has a representative in the atypical cytomata group. *Vide* Tables No. XIX and XX. Pages 61, 62.

But our difficulties do not end there. Because not to speak of the tissues, the cells themselves in most of the instances do not develop beyond **embryonic** stage, and therefore the elements of most of the tumours remain vegetative, *too rudimentary* and **undifferentiated** to determine their derivation, as they show no characteristic features of their parents. This makes the case of drawing out a genealogical table and to classify according to normal histology still more difficult. We have therefore no other way left but to classify this type of tumours according to the forms of their parenchyma cells and call them according to their *own* especial histology as they look or appear to us in their *form*. This makes the classification sometimes purely arbitrary or purely a matter of individual opinion. It can therefore be easily understood that in special cases how difficult sometimes it is to diagnose a case which would command universal acceptance.

Therefore according to the **shape** of these parenchyma cells a Sarcoma is classified as **round-celled** or **spindle-celled** Sarcoma. According to the **size** of these cells again

They do not form tissues and therefore it is not possible to classify them according to normal histology.

Even the cells do not help us to determine their integrity.

Diagnosis is sometimes impossible.

Sarcomata are classified according to the variations of their parenchyma cells.

such sarcomata are sub-divided into **small-round-celled-sarcoma**, and **large-round-celled-sarcoma**; and similarly especial terminology such as small-spindle-celled, and large-spindle-celled, are used to indicate the size of the parenchyma cells of those respective Sarcomata. The two varieties that is the small and large forms merge into each other without any distinctive measurement of size. According to the **arrangement** of the cells in relation to stroma and broad bands enclosing solid masses bearing resemblance to that of carcinomata it is sometimes described as **alveolar**. According to the lower degree of **anaplasia, organization** or development of these parenchyma cells into histological **tissues** resembling well-formed connective tissues it is described after those connective tissues corresponding to the non-malignant tumours; *e.g.*, osteo-sarcoma, chondro, or myxo, or fibro-sarcoma, etc. When all varieties of cells, such as the round-cells, and spindle-cells large or small, or both, are found together, in the same tumour such a mixture of cells in any single tumour is described as **mixed-celled-sarcoma** or **polymorphic-celled sarcoma**. When oat-shaped cells are found in a Sarcoma it is described as **oat-celled sarcoma**. With giant cells in it is called **giant-celled** Sarcoma. Myeloid cells are not sarcomatous. They are described as Myeloma or Giant-celled Benign tumours, and are not malignant. If malignant transformation takes place in a Myeloma it is described as Myelo-sarcoma.

Consistence.

**CONSISTENCE.**—According to the consistence, produced by the predominance of the matrix, or other changes such as degenerations, etc., a sarcoma is described as, **hard, soft, pulsating, medullary**, etc. The stroma sometimes undergoes mucinoid degeneration when it is called Myxo-sarcoma. Ossification or calcification may also occur by degenerations. It must be carefully noted that ossification, calcification, myxomatous change called myxo-sarcoma, etc., are degenerations. Fatty or mucinoid degenerations are apt to occur in the older portions of the tumour, which may ultimately become cystic. In soft

sarcoma, hæmorrhagic degeneration produced by extravasation of blood from a ruptured thrombosed vessel may also occur. Calcification occurs in the most chronic forms. Quiet necrosis as a result of hæmorrhage, or necrosis from deficiency in the formation of new vessels, failing to keep pace with rapid proliferation of the parenchyma, are other degenerative changes frequently seen in Sarcoma.

Malignancy of a Sarcoma depends upon the anaplasia of the cells constituting it. The more the condition of anaplasia the more undifferentiated, embryonic, and vegetative the cells remain, and the more malignant and virulent they are. The more they approach towards the formation of histogenetic tissues the more non-malignant they become. In a non-malignant tumour such as a fibroma or an osteoma the development of respective connective tissue cells are complete up to the adult standard. But in a Fibro-sarcoma or an Osteo-sarcoma it is only a tendency manifested by the sarcomatous cells to develop and **become organized** into **fibroid** or **osteoid** tissues which bear a close resemblance to the normal connective tissue fibres or bone cells. In some, according to the nature of its being less and less malignant, this phase of organization reaches a high degree of development **almost** into well formed adult type of fibrous or osteal tissue. The word *almost* should be noted. If the process were complete it would not have turned sarcomatous and malignant, but would have developed into a non-malignant tumour. These apparent histiomatoid growths range and occupy intermediate positions between the non-malignant connective tissue tumours on the one hand, and the malignant sarcoma on the other; and the degree of variation depends on the degree of the development of the sarcoma cells to a specialized adult type of connective tissue cell. In some tumours stages are differentiated and are described according to their condition as **fibrifying** or **chondrifying** stages and so forth. No definite line of demarcation can be drawn between the neighbouring modifications.

Degree of malignancy depends on anaplasia.

In a Sarcoma the type which is most virulent and malignant, develops *least* from the stage of embryonic form. The process of formation and its whole progress seem to be done **so hurriedly** that a round cell, for instance, which closely resembles a lymphocyte of the blood can not find time to grow further, but like war-time soldiers, are put into the field and made to functionate no sooner equipped. That is to say they at once divide and sub-divide. When inflammation takes place we find the same kind of connective tissue cells in a granulation tissue, called fibroblasts or osteoblasts, etc., which are also embryonic in construction and appearance. But these fibroblasts ultimately develop into normal fibrous tissue-cells or organize into bone through a stage of 'callus' formation; and no sooner the purpose is served, stop further, and incorporate as fully developed tissues with the other allied structures. In Sarcomata on the other hand a round cell remains small, and divide with limitless power of sub-divisions. It will not allow time to the stroma to develop properly for the supply of nutrition but just give a little interspace for the blood to pass. The result of the absence of such stroma is a formation which on naked-eye examination gives a peculiarly **homogeneous** appearance of the tumour, being due to no scaffolding to break the continuity of the parenchyma cells. Such small round cells may become large round cells, if they get time to do so, which in effect means the exhibition of less malignancy in the growth. Similarly spindle cells if growing rapidly would become less fusiform in shape, and may after passing through a stage known as oval, or oat-cells, even retrogress by anaplasia to round cells in character: Or all the varieties may remain mixed in the same tumour.

The growth  
and Metas-  
tasis.

The growth and extension of Sarcomata are peripheral and infiltrative like a carcinoma. The tumour cells proliferate irregularly along fascial planes and lymphatic spaces, leaving some interspaces for the blood to pass, with which they keep an intimate relation being mixed up with

blood cells; thus enjoying an uninterrupted opportunity of extension to the venous system. So that metastatic deposits occur both by lymphatic channels and the blood stream by embolism, as well as by permeation of the lymphatics also, although the latter form of dissemination is obscured by the more open door of exit through the blood vessels. Metastasis in Sarcomata is therefore mainly effected *via* blood vessels. And due to the extreme delicacy of these vessels extravasation, resulting from rupture especially after thrombosis of some larger veins, is very common in Sarcomata. A considerable area of tumour may thus undergo quiet necrosis as a result of such hæmorrhage.

The degenerations met with in sarcoma will be described more fully under individual tumours.

## SARCOMATA OF UNDIFFERENTIATED CELLS.

Sarcoma of Undifferentiated Cells.

**Pure Sarcomata:** that is those in which the constituent cells are of embryonic type, and are divided into the following groups, more or less artificially, according to the **size, shape, arrangement, character and site** of the constituent **cells** concerned. These are:—

(i) **ROUND-CELLED SARCOMATA.**—These may again be classified as, (a) small round-celled sarcomata, (b) large round-celled sarcomata.

(i) Round-celled Sarcomata.

(a) **Small round-celled Sarcoma** is the most malignant and most rapidly growing Sarcoma. This is also a blastoma of the most primitive type of cells. It may reach a great size. In consistency the tumour feels like granulation tissue, and is very soft. On gentle pressure the mass breaks down readily. In this condition they may be mistaken for cancer. They are pinkish white in colour, and the mass is very vascular. The growth usually spreads by embolism through the blood vessels, or by infiltration and direct inclusion. They do not as a rule affect the lymphatic glands, but not unfrequently the

(a) Small round-celled.

lymphatic glands are affected in this variety. Sometimes the vascularity is so abundant that the tumour pulsates.

Microscopically the tumour consists of small, round, nucleated masses of protoplasm loosely applied to one another with little intercellular substance. The nucleus is very definite, circular, or oval, having only a thin rim of protoplasm round the nucleus. It is sometimes situated eccentrically. The intercellular substance although slight is homogeneous in character. The vessels are thin-walled, like dilated capillaries.

The tumour cells are just slightly larger than the lymphocytes of the blood with which they closely resemble.

The very small size of the cells is regarded to be an indication of its high malignancy, since they divide and contribute hurriedly to the rapidity of the growth, and to the size of the tumour before they can themselves grow to a larger size. If they would find time to do so they would become less malignant, would allow the blood vessels to develop and organize more than mere endothelial tubes, would allow the formation of well marked stroma between the cells, and in which abundant protoplasm would encircle round the nucleus; and thus turn into tumours of the next variety known as Large round-celled Sarcoma to be described. The cells of the Small round-celled variety as stated above resemble the lymphocytes of the blood, or **just a little** larger than a normal lymphocyte, and in a particular specimen it is possible that many are actually lymphocytes which are mixed up with the tumour cells. Possibly the sarcomata cells represent the embryonic stage of **all** tissues, although it is commonly believed that the small round-celled type is derived from the fibrous connective tissue of the body.

The Sarcoma of this type exhibits retrograde changes to a marked degree and necrotic and hæmorrhagic changes are common.

Sites  
and age.

This type of Sarcoma may develop at any age; and any part of the body may be involved, the fascia of

muscles being particularly prone to give rise to it. They may originate from the skin, subcutaneous tissue, and periosteum. They are also found in mamma, testis, brain, muscles, and lymphatic glands.

Dissemination in this type is rapid and widespread, it occurs very early and proves always fatal in a few weeks. Operative interference are of no avail. Radium therapy yields encouraging results.

(b) **Large round-celled Sarcoma** is a less common form of sarcoma but having a wide distribution. In this variety the cells are bigger in size than those in the preceding form of Sarcoma. These cells contain one or two large oval nuclei, with a large and abundant quantity of cytoplasm around them, but the nuclei are clearer and less dense although they do not stain so deeply as in the small-celled form. The cells are not always spherical in shape and may even be polygonal, but **larger than any other kind of normal cells** of the body. The connective tissue stroma is interspersed between the cells, but it always assumes an embryonic type, and an alveolar arrangement is generally observed. Large Round-celled sarcoma is more firm, and more sharply defined than the small round-celled type. It is a highly malignant type although comparatively less so than the small round-celled type, as dissemination is not rapid and wide. They occur in the same sites of the body as the former type.

(b) Large  
round-  
celled  
Sarcoma.

As it possesses the power of certain degree of organization at all situations, it forms a definite tumour, but in the viscera it occurs as a **diffuse** growth.

Degenerative changes and the nature of dissemination are similar in character to the small round-celled variety.

(ii) **SPINDLE-CELLED SARCOMATA**.—Like round-celled variety, spindle-celled sarcomata are arbitrarily divided into **large** spindle-celled, and **small** spindle-celled types. The cells composing them resemble spindle-celled fibroblasts and connective tissue cells in granulation

(ii) Spindle-  
celled  
Sarcomata.

tissue, that is to say they are elongated and tapering at each end into a point like a spindle, which may be sometimes bifurcated at the extremities. Most of the anaplastic sarcomata belong to this group.

Spindle-celled sarcoma develops from connective tissue, fascia, aponeuroses, tendons, periosteum, etc. This form is one of the commonest variety of Sarcoma.

The cells and stroma of the spindle-celled sarcomata tend to arrange themselves in **whorls** as in fibromata, or in a somewhat fasciculated manner with the formation of a greater or less amount of intercellular substance. The organization of this connective tissue stroma varies in development and amount, namely, in some its development reaches **almost** into well-formed fibrous tissue which grows abundantly; in others quite reverse may be the condition. But this organization is always more abundant than in the round-celled sarcomata. The cells are packed together often in small bundles, and arrange themselves parallel to each other, in different planes forming a close interlacing felt work; the axes of which may sometimes cross each other, which on transverse section present the appearance of round cells with or without the inclusion of the nuclear part according as the section passes through the nucleus or misses it. In a flat section the nuclei are also found elongated and they appear not unlike the nuclei of unstriped muscle.

Types  
are:—

Spindle-celled Sarcomata present various types, according to: (a) the relative **sizes** of the parenchyma cells, (b) the different **shapes** of the parenchyma cells, (c) the **mixture** of the different varieties, (d) the development of the **stroma**, (e) the **degenerations**, and (f) **organizations** that might undergo in the substance of the tumour, viz. :—

- (a) Small-spindle-celled Sarcoma.
- (b) Large-spindle-celled Sarcoma.
- (c) Oat-celled Sarcoma.
- (d) Polymorphic-celled Sarcoma, or Mixed-celled Sarcoma.

(e) Fibro-Sarcomata or Recurrent Fibroid of Paget, etc.

Every one of the above varieties demands special and individual description.

(a) **Small spindle-celled sarcoma.**—Like the small type of the round-celled variety, this form of spindle-celled sarcoma grows rapidly attaining a considerable size and is the most malignant form amongst the spindle-celled ones. It is less succulent and therefore tend to form a somewhat firmer tumour than the small round-celled type. At first it constitutes of a localized growth of tolerably well defined character, although later on it invades and infiltrates the neighbouring areas but always keeping a sharply-defined margin. This form arises from fibrous connective tissue, fascia, tendon, and aponeuroses in any part of the body; in contrast to the larger type which is particularly associated with smooth muscles and periosteum; and when growing rapidly the cells may fail to reach the fusiform shape, and may divide no sooner formed, and thus proceed to degenerate into round-celled character. While turning into the latter condition they may pass through a stage known as oval or oat-shaped sarcoma to be described below. Sometimes a few multinucleated giant cells are also detected in Small spindle-celled Sarcoma which are hardly seen in the pure type of spindle-celled Sarcoma. The blood-vessels vary in amount, and are generally more developed than mere endothelial tubes which lie in intimately close contact with the cells of the tumour and sometimes seem to replace the vascular endothelium.

(b) **Large spindle-celled sarcoma.**—These sarcomata consist of relatively larger spindle-celled cells and grow from the smooth muscles and periosteum and sometimes from fibrous connective tissues. They also grow from the viscera. This larger variety is softer and of a deeper colour than the small spindle-celled type. In some instances, *e.g.*, the congenital sarcoma of

the kidney, which is a large round-celled growth some elements are muscle fibres, and the cells are transversely striated, when such tumours are described as Myosarcomata. For further particulars of blastomata from muscular origin *vide* Leiomyo-sarcoma.

(c) Oat-celled Sarcoma.

(c) **Oat-celled sarcomata** is a separate variety of spindle-celled sarcoma, and is so called on account of their cells being shorter and oat-shaped. This variety of sarcoma arises from fibrous tissue sheaths of muscles, and the periosteum, and sometimes from the connective tissue of the viscera.

Although the type is a highly malignant one, it yields to treatment more than the round-celled sarcomas. Degenerative changes are of similar character as in other varieties of Sarcomas.

(d) Mixed-celled Sarcoma.

(d) **Polymorphic-celled or mixed-celled sarcomata** are a mixture of round cells with small and large spindle cells. They are really composed of cells of various shapes, and sizes, retaining the characters of round and spindle-celled growths. Some adopt a distinct alveolar form, to be described. Polymorphic-celled Sarcoma commonly arises from the periosteum, but it has been found developing in the breast and uterus. Irregular multi-nucleate cells with deeply stained chromatin masses, which may be described as a type of giant cells, are frequently found in this mixed-celled type. Like the myeloids they may occur in bone, and the *confusion about the Myeloma is due to this type of Sarcoma* although they are seen in other situations also, but unlike them this mixed growth is highly malignant. The blood vessels in Mixed-celled Sarcoma are very rudimentary, and the inter-cellular substance is scanty.

Alveolar-spindle-celled Sarcoma.

(e) Fibro-Sarcoma.

(e) **Fibro-sarcomata or recurrent 'Fibroid' tumours of Paget.**—In some instances especially in the subcutaneous tissues, or sheaths of muscles especially of the rectus, organization of sarcomatous cells takes place transforming them into well-formed fibrous tissue.

When they reach so high a degree of development they remain on the border-line of malignancy. They grow slowly, rarely exhibit metastatic character and give rise to no secondary growths. From the start they exhibit the character of a less malignant nature. After operations they sometimes do not show any tendency to recur within two or three years; although the period of such intervals between successive operations becomes gradually less and less. Sometimes after as many as half a dozen operations a tumour of this nature may turn malignant as an ordinary spindle-celled sarcoma. It is often described as Chronic Fibro-Sarcoma and usually occurs in the abdominal wall of young women as a firm, round or ovoid swelling which is fixed to the abdominal muscles especially when they are in action. The tumour is otherwise movable, which can be demonstrated if the patient lies flat and relaxes the abdomen.

### ALVEOLAR SARCOMA.

The Alveolar Sarcoma is not a particular form of sarcoma but in this type there is an attempt of grouping of cells especially of spindle-celled type in **alveolar** form. The difference lies in the arrangement and not in morphological appearance. The cells are grouped in alveoli composed of bands of fibrous tissue. But this chamber-like arrangement for a group of cells is the characteristic feature of Carcinoma and not of Sarcoma, as already described. Moreover the constituent cells are often found of uniform size, and some are neither like the round, nor the spindle-shaped cells, described above; but resemble the rounded or ovoid cells of epitheloid character. They are of course not cubical or columnar in shape as in carcinoma. In some there is no intercellular substance between the cells the existence of which is a characteristic feature of sarcomata. Another peculiar feature observed in some alveolar form is that like the Melanotic Sar-

Alveolar  
Sarcoma.

comata they often arise from the skin, and are generally very malignant.

Some alveolar growths are carcinomata, some are endotheliomata, and some are sarcomata.

Some of these so called alveolar growths are really carcinomata, and possibly the majority are endotheliomata, wrongly diagnosed. By carefully pencilling them with a camel's hair brush if a delicate intercellular stroma can be demonstrated those cases are evidently proved to be sarcomata.

Organized  
Sarcomata.

## ORGANIZED SARCOMATA

or

### SARCOMATA OF DIFFERENTIATED CELLS.

From Sarcomata consisting of embryonic elements and therefore most malignant, we now turn to those which attempt to organize into *tissues* which may be differentiated into groups according to the predominance of the tissues that can be identified in them and therefore showing a tendency to non-malignancy. Generally the following types are met with, exhibiting various intermediate stages according to the degree of malignancy, *viz.*, Fibro-sarcoma, Lipo-sarcoma, Myxo-sarcoma, Leiomyo-sarcoma, Lympho-sarcoma, Chondro-sarcoma, Osteio-sarcoma, Glio-sarcoma, Neuro-sarcoma.

These more or less organized Sarcomata should not be confused with Mixed-celled Sarcoma. The histioid tumours are constituted as the result of organization of the parenchyma cells into fibroid, lipoid, myxomatous, lymphoid, myeloid, chondroid, osteioid, gliomatous, and neuromatous tissues, that give rise to the types mentioned above. The prefixes are used to indicate the particular kind of connective tissue to which the pure sarcomatous elements attempt to develop; *e.g.*, in Osteio-sarcoma there is an apparent development of bony tissue to the naked eye and touch, but no true bone is produced. Only the intercellular substance becomes infiltrated with materials like that found in the matrix of cartilage, which are afterwards impregnated with calcium salts.





LYMPHO-SARCOMA.

Some of the above types demand description in greater details which follows:—

(1) **LYMPHO-SARCOMA.**

(1) **Lympho-Sarcoma.**

In some of the lymphatic glands a type of sarcoma is met with called **Lympho-Sarcoma**. This is a variety of sarcoma in which the intercellular substance is of a delicate reticular nature, resembling the retiform tissue found in the normal lymphatic glands, or lymphadenoid tissue. The tumour looks like lymphoid tissue in appearance and is also derived from lymphoid tissue. It has a structure consisting of very small round cells set in a reticulated stroma well supplied with delicate capillary vessels but exhibiting no distinction between cortex and medulla. The cells appear identical with the ordinary lymphocytes; and the sarcomatous nature of the cells is evidenced more by the clinical features, than by their histological characters. Lympho-sarcoma originates in a pre-existing glandular tissue, such as the glands at the root of the neck, tonsils, (*vide* photo-plate No. LI) thymus, mediastinum, Peyer's patches, cæcum, etc. Three of these situations, *viz.*, the tonsils, the mediastinum and the cæcum are affected in the majority of the instances. It is a peculiar feature why it has not a wider distribution as might be expected from glands all throughout the body. It grows as a rapidly developing tumour which is at first painless, firm, and elastic, later becoming tender and painful, due to implication of nerves. Adhesions to surrounding parts very soon takes place by infiltration, and secondary growths quickly appear at the neighbouring glands; this latter feature of lymphadenoid dissemination is very characteristic of lympho-sarcoma. The covering skin, with the progress of the growth, becomes shiny and congested exhibiting a network of dilated veins. If the growth bursts into ulcer it rapidly grows into a bleeding fungating mass exhibiting a rounded

and irregular surface. In most of the instances the secondary growths develop more rapidly than the primary one.

Lympho-sarcoma is the **most** malignant form of sarcoma although the cells organize themselves very near a histiogenetic tissue. Death generally takes place by extensive visceral metastases.

Two clinical examples deserve some further description, *viz.* :—

Mediastinal  
Lympho-  
Sarcoma.

**Mediastinal Lympho-Sarcoma**,—occurs in the mediastinum from any lymphoid tissue in the thoracic cavity, namely, the pulmonary or bronchial glands, remains of the thymus, etc.

Mediastinal Lympho-sarcoma attains a great size invading the lungs and even spreads to the lymph glands of the abdomen. The tumour is beyond the scope of practical operative surgery.

Intestinal  
Lympho-  
Sarcoma.

**Intestinal lympho-sarcoma**—arises like multiple nodes and together with the adjacent glands forms a large mass. The growth extends underneath the mucous membrane and does not as a rule cause any ulceration. Multiple nodes often appear in the walls of the gut. In the later stages extension takes place into the surrounding connective tissues and the peritoneal covering.

The tumour is amenable to operative measures and sufficiently wide extirpation offers a reasonable chance of success.

It is worth remembering that Leukæmia, which consists of changes in the blood and lymph glands, the non-malignant Lymphoma, and the malignant Lympho-sarcoma are often associated with each other so closely that mistake is always possible to be committed in the diagnosis of any of these conditions. In some clinical instances Mediastinal Lympho-sarcoma was determined only after autopsy, in the cases which exhibited the blood picture and clinical features of lymphatic leukæmia; demonstrating the relationship and co-operation of these

blastomatoid and blastomic conditions. *Vide* Chap. I. Blastomatoid and Neoblastic nature of Leukæmia. P. 35.

## (2) LIPO-SARCOMA.

(2) Lipo-Sarcoma.

**Lipo-sarcoma** is a true blastoma which is derived from adipose tissue. We have described under Lipoma the scantiness of capsule in such a tumour. It has also been stated that in some instances of Lipoma the cells may turn embryonic in type and thus set in malignancy transforming a Lipoma into Lipo-sarcoma. In such instances no capsule can be distinguished.

It must be understood at the very outset that fatty degeneration may occur in any Sarcoma and the discovery of fat cells in any particular section of a tumour should not be hurriedly declared to be a part of a Lipo-sarcoma. It is not a degeneration.

Lipo-sarcoma may arise from malignant transformation of a non-malignant Lipoma, or may do so independently and directly from normal fat cells of the body.

The tumour is composed of cells of an embryonic type containing fat globules in their cytoplasm. Like Lipoma the fat globules present the appearance of so many vacuoles in a section, the fat being dissolved out by xylol leaving the spaces empty in a paraffin section. It is not possible in a very malignant form of Lipo-sarcoma to determine the true character of the cells as they may not organize sufficiently to be differentiated as lipomatous cells.

## (3) FIBRO-SARCOMA.

(3) Fibro-Sarcoma.

**Fibro-sarcoma** is the least malignant tumour in the Sarcoma group. It develops slowly into a firm tumour. Metastasis does not occur, especially at the beginning. The cells constituting them are larger than those in a non-malignant Fibroma, but are more irregular in shape. Abundant matrix sometimes develops in this

type of malignant fibrous tumour. This is a class of tumour which serves as a good example to demonstrate that according to anaplasia malignancy increases. With increased malignancy the cells become more or less spindle-celled in their appearance. It sometimes becomes a question of great difficulty to distinguish between the cells of non-malignant fibroma, malignant fibro-sarcoma and some conditions of fibromatosis which are blastomatoid in nature.

Fibro-sarcomata of the breast usually develop into massive tumours having a comparatively low grade of malignancy. They may both be solid, and cystic as occurs in Brodie's sarcoma described below.

Brodie's  
Sero-cystic  
Sarcoma.

Occasionally intra-canalicular form of fibro-adenoma of the breast grows to an enormous size, and in them sometimes the connective tissue may assume sarcomatous character, when they are described as Brodie's **serocystic sarcoma**.

From practical point of view the malignant character of a fibrous tumour must be realized, and so long that is ascertained it matters very little from treatment point of view whether such a tumour is a sarcoma or a fibro-sarcoma.

(4) Osteo-  
Sarcoma.

#### (4) OSTEO-SARCOMA.

**Osteo-sarcoma** tends to produce bone, and according to the degree of success in the formation of bone various grades may be observed between non-malignant osteoma and various fibro,—or spindle-celled sarcoma to the stage of Osteo-sarcoma. Such infiltration of calcium salts in the fibrous tissues of a tumour ultimately completing into bony tissue may not be uniform throughout the tumour. The constituent cells of an Osteo-sarcoma exhibit various pictures. In the majority of the cases they exhibit the character of polymorphic cells. Surrounding the bone trabeculæ the cells arrange themselves and behave like osteoblasts. In some tumors dis-



OSTEO-SARCOMA.



tinct cartilage cells are found which are described as **Osteo-Chondro-Sarcoma**. Various other anomalies and gradations in bone formation may be observed. Starting from true bone formation occupying a great part of the tumour mass, or only in islands of bony tissue at different isolated places which are discovered by making thin sections, to small spicules incorporated in the substance of the tumour are observed. But diagnosis becomes difficult from calcareous degenerations in a slow growing fibro,—or chondro-sarcoma. In some instances the bony organization may not reach beyond the *osteoid* stage consisting of trabeculæ but with little or no deposition of calcium salts in the matrix. Such anaplastic types of Osteo-sarcoma grows more rapidly from metastases and are comparatively most malignant of all the other variations. The more organized they are the more localized they remain as a slow growing, hard, comparatively less malignant tumour without showing any evidence of metastasis; till many instances of similar nature often remain entirely localized. *Vide* photo-plate No. LII. When metastases in Osteo-sarcoma occur it may reproduce bony sarcoma or pure sarcoma.

For further details *vide* chapters on the diseases of bones.

#### (5) **CHONDRO-SARCOMA.**

(5, ' chondro-Sarcoma.

As the name indicates **chondro-sarcoma** is derived from cartilage. As in the non-malignant chondromata it arises only in connection with those parts where cartilage is normally present or where it occurs in connection with bone. Presence of cartilage-cells in other situations is to be regarded as **teratomatous**.

Like Osteo-sarcoma the structure of Chondro-sarcoma varies with the degree of malignancy, and the more anaplastic the cells are the more malignant the growth becomes. In the more organized and slow growing forms it is only by an examination of the growing margin of

the tumour one can definitely make a diagnosis; where a distinct zone of cellular tissue which is sarcomatous-looking can be made out. In the comparatively more malignant growths the cartilage cells appear to be imperfect, irregular, and indistinct, but the sarcomatous or embryonic elements predominate.

Clinically Chondro-sarcomata develop into massive tumours closely resembling their non-malignant representatives.

Mucinoid degeneration is very common.

Mucinoid degeneration is especially very common in Chondro-sarcoma. As a rule Chondro-sarcomata are much more liable to retrogressive degenerations than other forms of sarcomata of differentiated cells. In most of the instances no cartilage cells may be differentiated over considerable area, and only an excess of myxomatous matrix is seen.

Chondro-sarcomata do not usually disseminate widely, although they are locally very destructive, where they infiltrate and destroy the adjacent tissues. As a rule Chondro-sarcoma does not grow very rapidly.

(6) Leiomyosarcoma.

#### (6) LEIOMYOSARCOMA.

**Leiomyosarcoma** is a tumour which is derived from smooth muscles, but for practical purposes it is not often possible to ascertain that. In alimentary canal such an instance has been found. It can be distinguished by the arrangement of their cells and their nuclei. The nuclei are often scanty as compared with spindle-celled sarcoma, and they are longer with rounded ends. The cells do not so closely interlace like felt as happens in spindle-celled sarcoma, but are separated by distinct connective tissue stroma in which nutrient vessels pass. It is with difficulty sometimes longitudinal striations in the cells may be made out.

It is a very malignant tumour, metastases taking place in the viscera and lymph glands.

(7) **RHABDO-MYOSARCOMA.**

(7) Rhabdo-  
myo-  
Sarcoma.

**Rhabdo-miosarcomata** are malignant growths of striated muscles, and are very rare although comparatively more common than their non-malignant representative. They are occasionally met with in the genito-urinary tract. Teratoma of this nature is more common than blastoma of this kind.

(8) **GLIO-SARCOMA.**

(8) Glio-  
Sarcoma.

**Glio-sarcoma** is a malignant form of transformation of Glioma. Its extreme cellular nature with few fibrils are features which give enough indication to establish the diagnosis.

(9) **NEURO-SARCOMA.**

(9) Neuro-  
Sarcoma.

**Neuro-sarcomata** are malignant growths of embryonic nerve cells or neuroblastomata. From retina and adrenal certain type of malignant growths are derived which grow rapidly and form widespread metastases. Both macroscopically and microscopically they appear very like round-celled sarcoma. These tumours are usually described as Glio-sarcomata.

(10) **MYXO-SARCOMA.**

(10) Myxo-  
Sarcoma.

**Myxo-sarcoma** as it is usually known, in the majority of the instances are only mucoid degeneration in a sarcoma. True malignant blastoma composed of myxomatous tissue whose cells have the power of secreting mucus are very rarely seen.

True Myxo-sarcoma is a very highly malignant tumour in which metastases occur very early.

III. PIGMENTED TUMOURS.

III. Pig-  
mented  
tumours.

Pigmented tumours such as Moles and Melanomata are tumours constituted of specific **nævus** cells which

### III. Pigmented tumours. Pigment melanin.

#### Source.

contain variable amount of a special pigment called **melanin**. This melanin is an **iron-free** pigment which indicates that it is not derived from hæmoglobin of the blood. On the other hand this pigment contains a large amount of **sulphur**. The source of this sulphur perhaps comes from the protein molecule or amino-acids containing sulphur, such as those of cystin group.

Pigmented tumours are a special class. Their cells are specified called Nævus cells.

A Nævus may be Pigmented or Non-pigmented.

Pigmented races do not suffer from this kind of tumour.

The tumour as believed before is not a sarcoma and therefore the designation Melanotic Sarcoma ought to be discarded. The *origin, nature,* and histological structure are so varied that this kind of tumours are grouped under a separate classification called **pigmented** tumours, and the specific cells are called the **nævus cells**. We know that the deepest layers of the epidermis naturally contain pigment in dark races, and the nævus cells which constitute the malignant and the non-malignant pigmented growths bear a close relationship to the basilar layers of epidermis and to such derivatives of epidermal epithelial cells such as hair follicles and sweat glands. The commonest and the most well-known example of this condition is found in the congenital **mole** or the **nævus**. It is interesting to observe from the very beginning that a nævus *may be pigmented or non-pigmented*, although the latter condition is rarer; and therefore we should not depend upon the presence of pigment alone in our diagnosis of a tumour of this kind. On the other hand it is still more interesting and curious that black or coloured or pigmented races never suffer from this kind of growth. The statement may be too sweeping.

The **nævus** cells are specific cells, and in the congenital moles different stages of the development and segregation of the cells of the epidermis converted into those of the tumour may be demonstrated; exhibiting not only an intimate relationship with the epidermis but also an insensible gradation of transformation from the one into the other. These observations distinctly prove that the tumour is epithelial in origin. Moreover its arrange-

ment of cells in alveolar form is another strong argument. But there are instances proving the development of some of the tumours from the special mesoblastic pigments known as melanoblasts or **chromatophores**, which indicate that the origin may be of varied nature. It may be noted that the neoplastic chromatophores do not actually produce melanin. Some tumours therefore have one pedigree or mode of origin, whilst others have another.

Microscopically a nævus cell is a rounded or spheroidal cell usually pigmented, the amount of which is by no means constant, but the majority of cells under microscope exhibit a condition of being loaded with golden brown granules, or only a few cells at different areas of the sliding stage may be found to contain them. It may be possible that nævus cells are true **melanoblasts** of specific type and not mere epiblastic cells working as carriers of pigment being especially drilled and mobilized for the purpose of holding only a special charge for the time being. Unna believes that they are epithelial in nature, being originated from the pigmented cells of the rete Malpighii (the deepest layers of the epidermis).

Histology  
of the  
Nævus  
cells.

Two main varieties of Pigmented tumors are described; viz. :—

Two main  
varieties.

A. The Non-malignant form or the **moles**.

B. The Malignant forms or the **melanomata**. These again are recognized in two separate forms; viz. :—

(i) **Melanotic Carcinoma**, that is those arising from the rete Malpighii of the epidermis. Often described as Nævo-carcinoma or Melano-carcinoma.

(ii) **Melanotic Sarcoma**, that is those arising from connective tissue chromatophores.

For the convenience of description we shall describe the Non-malignant types of Pigmented tumours here instead of in the chapter for other non-malignant tumours.

Non-malignant pigmented tumours. The Nævus or Mole.

## NON-MALIGNANT PIGMENTED TUMOURS.

### THE NÆVUS or MOLE.

The Nævus or Mole is a very common wart-like congenital abnormality of the skin. It is usually found on the face, the neck, arm, or the back although it may occur at any part of the body. It may be markedly raised from the surface some resting almost on a stalk-like process, or may be almost flat and level with it. It may be pigmented, which is more common, or non-pigmented. A pigmented Nævus may be grey, brown, or black in colour. Often one or more coarse hairs of unusual length in contrast to those of the neighbourhood may be seen on the surface, apex, or margin of a Nævus.

Two forms.

Two forms of Moles are met with, *viz.* :—

(a) Infantile form.

(b) Adult form.

(a) Infantile form.

(a) The **infantile** form.—This is the one met with as congenital mole of infants and children. In this form the cells of the basilar layers of the epidermis, or of the hair follicles and sweat glands become swollen and isolated which form clumps in the underlying tissue. The cells pass through three stages in sequence: *viz.*, the stage of proliferation with pigmentation, followed by depigmentation and then by quiescence. The most important surgical point is that any injudicious or incomplete operative or semi-operative surgical interference may at any time induce the most virulent type of malignancy.

Important Surgical point.

(b) Adult form.

(b) In the **adult** form the epidermis sends elongated processes down into the corium, and in the intervening papillæ the clear rounded pigmented cells group into an alveolar arrangement lying in a fairly dense fibrous tissue stroma. Usually no connection with the epidermis can be seen and the exhibition is one of quiescence.

As a rule great majority of moles remain non-malignant sometimes growing slowly for a long period and then

becoming quiescent ultimately undergoing fibrotic changes. Otherwise any irritation or trauma is a source of grave danger, as such a mole may at any time develop malignant characteristics.

A pigmented mole should be considered as a non-malignant Melanoma, and it may assume an active growth even without any irritation; the growth in that circumstance becomes more cellular, the cells also exhibiting an unusual activity.

**Clinically**,—as a rule this form of cutaneous non-malignant Melanoma develops from a pre-existing congenital mole. In threatening cases at first a patch, deeply pigmented, appears round a mole which may extend several square inches over the skin. The pigmentation passes to the deeper layer of cutis vera otherwise no induration or infiltration are observed in a section under microscope. Later a distinct tumour develops at the centre of this pigmented area. The growth does not increase rapidly. Old cases may at any time exhibit signs of dissemination. Melanotic Whitlow may develop as a simple "black spot" under the thumb nail which may remain quiescent for even four or five years; the size remaining as small as a pin's head. This "black spot" may suddenly develop into a small wart-like tumour. No involvement of lymph channels or glands may at first be apparently detected by a superficial clinical examination, although they may be permeated to several inches by the Nævus cells. Thus when malignancy sets in, it takes to the **most** virulent type of malignant neoplasm ever known.

Clinical features.

Melanotic whitlow.

Apart from pre-existing moles pigmented tumours may develop from trauma such as injury on the foot occurring from a rusty nail. (Kettle.)

**TREATMENT**.—Thorough excision and extirpation of the whole affected area and a good part of the normal tissue suspected of lymphatic permeation is the best method to adopt. No trace of any pigment cell should

Treatment.

be left behind which may be traced even to a small lymphatic, as that is sure to excite recurrence. In digits amputation done early may save the patient's life. Radium treatment is of some avail in unapproachable parts of the body.

Important  
Surgical  
point.

It is always safe and best never to interfere with a Mole in the quiescent condition. Least suspicion arising from their nature, drastic operations as stated above with careful tracing and extirpation of all lymphatics must be undertaken as early as possible. When malignancy starts a very simple looking little spot may be traced along several inches or even feet of a single lymphatic channel.

Melanomata  
or Malignant  
Pigmented  
Tumours.

## MELANOMATA

or

### MALIGNANT PIGMENTED TUMOURS.

Pigmented tumours of malignant nature are described as **melanomata**.

Melanomata arise from those portions of the body where **pigment naturally exists**. The pigment as described above is **melanin**, and physiologically it is the sub-cutaneous tissue and the choroid coat of the eye where such pigments exist. Melanoma is a neoplasm in which this pigment constitutes the main factor. A Mole on the other hand may not be pigmented at all. We have already described how a quiescent non-malignant Mole at times exhibits activity by the proliferation of these particular type of pigment cells containing melanin pigments, especially described as **nævus cells**. Such nævus cells are found in a Melanotic Sarcoma which may be composed entirely of spindle-cells, but exhibiting three prominent peculiarities, *viz*; (a) the cells may show the nature of **epitheloid** character; (b) they sometimes arrange in **alveolar** form; and (c) they are heavily pigmented. Such instances are seen to grow from the choroid coat of the eye; and when they do so they are usually entirely composed of spindle-cells, all of which contain melanin. This

variety is described as **melanotic sarcoma** and the especial peculiarity of the sarcomatous type of Melanoma is that it is heavily pigmented. Those pigmented tumours which arise from the skin on the other hand, such as from moles or nævi or birthmarks, as described under *Angeiomata*, *vide* above, the epitheloid cells are, (a) spheroidal or polygonal in type; (b) they arrange themselves in alveolar form as a rule; and (c) they contain only a few scattered granules of pigments. For this epitheloid nature and their frank alveolar arrangement in these tumours, as well as their very early metastases through lymphatics Unna described them as **melanotic carcinoma**; although an interstitial substance can be demonstrated between the cells, a feature which does not exist in carcinoma. Pigmented neoplasm of pure epitheliomatous type is never seen; but tumours of this nature are composed of **nævus** cells the origin of which has been discussed above. The especial peculiarity of the carcinomatous type is that the pigments are only scattered. Clinically it is immaterial to differentiate between the two species because both are equally killing to the host. It appears that all Melanomata belong to one and the same class of new growths, which frequently exhibits all stages between the two types. We shall therefore describe both the forms under one heading of **melanoma**. But as a rule both carcinomatous and sarcomatous forms may be found in the cutaneous type, and those arising from the choroid of the eye are distinctly sarcomatous.

Melanomata are found in the following situations of the body, *viz.*:— Melanomata.

Moles, arising primarily anywhere in the body:

Eyes—arising primarily from the Choroid and Iris.

Liver, as a rule secondary.

Adrenal, may originate primarily.

Neoplastic warts and moles around the Umbilicus.

Ovary and breast, may originate primarily.

Meninges and brain, may be primary.

Anus, arising primarily.

Trauma, *e.g.*, in the foot from rusty nails in the boot. Rarely in the tonsils secondarily.

Appendix, nævus-like growth is sometimes found occurring in its mucous membrane as spheroidal carcinoma.

It will help the beginner to note the first alphabets of the above lines, and remember that Melanomata arise in **m e l a n o m a t a**. They usually arise from a pre-existing mole; and sometimes in the iris and choroid, but not so common in other positions mentioned above.

Melanomata are **exceedingly malignant**. In fact they surpass all other known forms of malignant neoplasms in their character of malignancy. It is really the most virulent form of malignant disease, as metastases take place in the lymphatics and the liver as well as in other internal organ at a very early date, even when the primary growth may still be of an insignificant size. The choroid type may show delayed metastasis, it is a sarcomatous melanoma, and it has an especial tendency to settle in the liver.

Naked-eye  
appearance.

To the naked eye the primary tumour may be a small insignificant spot without exhibiting any occasion of serious clinical disturbance. This spot may be greyish in colour or of blackish brown or slaty hue. Some portions of the growths, primary or secondary, may be quite colourless while the adjacent portions may be quite black.

Micros-  
copical ap-  
pearance.

Microscopically the melanin granules are found to be peculiarly distributed, *e.g.*, some lying in the stroma between the alveoli, some inside the parenchyma cells. At places they may be evenly distributed, at others again they may show themselves in clumps or in masses. There may be a condition of general deposition on the skin of the other parts of the body of the patient when such features are clinically described as **melanosis**. In a patient suffering from Melanoma, nævus cells or melanin may be detected in the urine when it is described as **melanuria**. The pigment appears under microscope as

Melanosis.

Melanuria.

amorphous granules of irregular shapes varying in size from the finest particles to masses as large as the cells themselves. They look pale brown in colour or appear in sepia shade, as we find in photograph, and are generally situated in the intra-cellular spaces. The constituent cells may be spindle-shaped or spheroidal or polygonal having an alveolar arrangement. Sometimes definite adenomatous or acinous formation may be observed.

Most important feature in Melanomata is their size and metastases. The size of the primary Melanoma is not very large, and it seldom attains a great size; the secondary growths also are more characterized by their number rather than by their size. It is the widespread metastases which turns the condition fatal. Metastases soon takes place in the nearest lymphatic gland spreading to the deeper tissues, and then to the organs of the whole body, which become affected by general melanosis by blood stream. Liver, lungs, brain, kidney are soon affected by widespread secondary growths. Even when malignancy sets in, in many cases a period of quiescence is sometimes observed, and in some cases temporary immunity is also sometimes seen. The dissemination occurs not only by the lymphatics but also by the blood stream from the beginning, and the early invasion of the liver, lungs, kidney and brain is the result of blood infection; when melanin also appears in the urine; and in the blood, described as **melanæmia**.

CLINICALLY,—the tumour by itself may be only an insignificant spot which may cause no anxiety or clinical disturbance. Unless the melanosis on the surface is pronounced the secondary growths also may not cause any trouble for sometime. Usually some brown or black nodules may develop. Visceral metastases may give rise to visceral symptoms masking the actual nature of the fatal condition. The general health is remarkably little affected, and even when it becomes virulently malignant the patient may yet remain robust; or the condition may be interrupted by quiescent intervals or temporary

immunity; during which periods even many of the melanotic nodules caused by melanosis may treacherously disappear, which after sometime may reappear to hurriedly carry away the patient.

Diagnosis.

DIAGNOSIS, rests upon the detection of the peculiar cells. It is the type and arrangement of the cells and not the pigmentation which matter, and on the determination of the former the correct diagnosis depends. It must be remembered that tumours with hæmorrhagic foci are likewise pigmented; they are not sulphur granules, but iron pigments.

Main  
Surgical  
point.

MAIN surgical point is to take drastic measures immediately and as soon as diagnosis is established. Not only the lymphatic glands but the lymphatics also must be followed by linear incisions and thoroughly extirpated. The digit should be amputated in a Melanotic whitlow, and the lymphatics traced and extirpated; or the affected limb should be amputated if any doubt remains in the successes of such extirpation of the lymphatics. Unapproachable parts may be treated with radium.

IV. Hyper-  
nephro-  
mata.

#### IV. HYPERNEPHROMATA OR ADRENAL TUMOUR OF GRAWITZ.

Hypernephromata is a small class of tumours, which as the name implies, arise from the suprarenal substance, and chiefly grow at the upper half of the kidney below its cortex. It is a malignant tumour as it ultimately kills the host, and metastases take place at the usual sites which we describe as the Lands of four Ls; viz.:—

Lungs.

Long bones.

Liver.

Lymphatic glands.

Its peculiar  
features.

ITS PECULIAR FEATURES.—But the most peculiar features of its malignancy are that; (i) the tumours have no special tendency to invade the lymphatic glands; (ii) the duration of life after the

onset extends to five years; (iii) metastases take place very late in the lungs, long bones, and the liver; (iv) great majority of them occur in the kidney, and it is unusual or extremely rare to find them elsewhere; (v) moreover this form represents seventy-five per cent. of all renal growths, and therefore is the commonest type of renal tumour; (vi) although malignant at last it has a capsule of kidney substance which it shows no tendency to destroy or break through for a number of years, till which time it remains encapsuled as a locally invading growth without producing any metastases; or remain even sharply separated from the kidney substance by a definite fibrous capsule,—a feature unknown in true malignant blastomata, —and in that respect giving a strong evidence as a support for those who invest it with the honour of non-malignancy.

ORIGIN.—The origin of Hypernephromata was a Its origin. hotly disputed question. Dispute arises in including some Adeno-carcinomata and some Teratomata or Embryomata in the group; but there remains a well-defined group which is entitled to be called after its first observer Grawitz.

Grawitz's Hypernephromata arise either in the suprarenal gland, or in the isolated 'rests' of adrenal substance lying concealed under the cortex. And for all diagnostic purposes Boyd's Law may be followed with Boyd's Law. practically cent. per cent. success; which is as follows; **"if the adrenal cannot be found the tumour is pretty certain to be a hypernephroma."**

ITS GROSS APPEARANCE.—For all practical Its gross appearance. purposes it may be assumed that when the surface Cut pomegranate. of a section of a kidney tumour presents an appearance like a half-cut Indian pomegranate preserved at Kabuliwalla's shop for sometime, it is possibly a Hypernephroma. It is possible in most of the instances to make a correct diagnosis from a macroscopic examination only. It forms a large rounded encapsuled

"The  
Golden  
Tumour."

mass which projects from beneath the cortex of the kidney usually from the upper half with a bossy surface of its own, causing comparatively little change in the general shape of the kidney. On section it presents many cysts of varied sizes and colours exhibiting, something like a cut pomegranate, some isolated portions of which have started to become stale or brown. That is to say it exhibits many cysts surrounded by striated semi-transparent septa of fibrous tissue which gives it a mesh-like and remarkably variegated appearance, containing many areas of rounded and localized spaces of red, and maroon and some of peculiar golden yellow colour. The latter features give it a special name known as "**The Golden Tumour.**" All these variations of colour and consistence of the spaces are due mainly to: (i) hæmorrhage (red); (ii) accumulation of a special fatty substance of lipoid nature which is an ester of cholesterin and glycogen granules. These fatty or lipoid substances give it a foamy appearance under microscope; the spaces becoming vacant and prominent by the fat being dissolved out during preparation; (iii) cystic degeneration; (iv) mucoid degeneration; (v) necroses.

Other  
malignant  
kidney  
tumours  
are white  
or brain-  
like in  
colour.

It must be remembered that *the colour of other malignant tumours of the kidney is white or whitish and brain-like*. And the more brain-like the isolated areas are the more it is possible to be carcinomatous or adenocarcinomatous or sarcomatous in nature. This explains that a large proportion of the kidney tumours which used to be regarded as Hypernephromata are in reality adenocarcinomata arising from the renal tubules.

Micros-  
copical  
features.

MICROSCOPICALLY,—the adrenal Hypernephroma resembles many of the structures of the suprarenal body. A distinct zone of fasciculata of the suprarenal gland consisting of densely packed columns of polyhedral cells containing glycogen are observed. But the picture is not so constant. In some the zona glomerulosa or the zona fascicularis is reproduced more or less faithfully. The cells may be grouped in either solid alveolar or in

trabecular arrangement, or sometimes scattered quite diffusely; in both types they are separated by an extremely abundant capillary net work, which renders extravasation of blood to take place frequently. The cells themselves are characteristic. They are slightly larger than the ordinary renal cells, and appear granular, owing to the presence of the lipoid and glycogenic granules. Sometimes pigment granules due to disintegration of hæmorrhage may be detected.

**CLINICAL FEATURES.**—In consideration of the gravity of the lesion renal Hypernephromata give rise to very few symptoms. The disease occurs at any age between forty-five to sixty. The tumour grows to a considerable size, and the only symptom complained of being *recurrent* attacks of hæmaturia, often after some unusual exertion. But the hæmaturia never becomes continuous which is a characteristic feature of carcinoma and sarcoma of adults. There is a constant complaint of marked pain in the back, of the nature of backache. The progress of growth is slow, and the duration of life extends generally up to five years. Metastases are prone to appear in the lungs and long bones, or rarely in the lymphatic glands; and often the first clinical symptoms are manifested by a bony growth which is usually diagnosed as a primary osteal sarcoma. Clinical features.

**TREATMENT**—consists in Nephrectomy, for the details of which the reader is referred to regional surgery. Treatment.

This finishes our description of practically all common tumours which are known as **autochthonous** Blastomata; that is to say the tumours which are derived from **unipotential** cells, originally indigenous of the soil; or in other words native to the host, or the individual bearing them. Autochthonous blastomata.  
Unipotential cells.

Some autochthonous blastomata may be **pluripotential** in nature, that is to say constituted of many **kinds** of cells; and therefore described as **mixed-tumours**. These tumours are formed of many tissues of varied structures Pluripotential cells.  
Mixed tumours.

such as cartilage, fibrous tissue, epithelial elements; which however do not form any recognizable fœtal organs.

Heterochthonous blastoma. Chorion epithelioma.

The term autochthonous means something derived from the individual *itself*. And unipotential cells are those which are potent or have power to form only **one** variety of tissue. Practically all Blastomata are autochthonous, excepting only one, originating in the pregnant uterus after abortion called **chorion-epithelioma**, is **heterochthonous**; that is to say the cells constituting the tumour are derived from foreign cells not belonging to the host, and that in this case is originated from the fœtus. In this sense it is an **embryoma**, but not an **organ** tumour, as it is composed of **cells**, and not even of tissues, not to speak of organs. It is therefore a **malignant cytoma** or a cell tumour. It is sometimes

Teratomata.

described under **teratomata** on the ground of its originating from cells derived from a foreign individual. All experimental tumours produced on lower animals in the laboratory are tumours of this class. But in the face of rarer instances, as Chorion-epithelioma has been observed not only in the uterus not associated with pregnancy but also in the **ovary** and even in the **testicle** of the male, and in some more rarer instances in the thorax, it is safer to follow Adami's nomenclature and describe them under **teratoma** and **teratoid** tumours, which follows.

Teratoids.

## TERATOMATA OR EMBRYOMATA AND TERATOIDS.

Teratomata or Organomata.

**TERATOMATA**—are tumours consisting of different tissues or organs derived from cells originating from all three primitive layers of blastoderm; and which are derived from another individual that is **heterochthonous** in nature, or may be from the same individual that is **autochthonous**. But as they are capable of giving rise to **all the tissues of the body representative of all three**

primitive layers of blastoderm, their cells are called **totipotential** cells, and the tumours are described as **Embryomata** or **Organomata**.

The origin of a Teratoma may be traced from the inclusion of an additional embryonic cells as the result of extra-fecundation or some other abnormal anomaly of fecundation, all the suggested theories of which will be described presently. Included embryos, or portions of a second embryo, may remain completely buried in a rudimentary condition for a considerable number of years in the tissues of the host, and suddenly multiply at any time of the life of the patient and give rise to a neoplastic growth, sufficiently developed to be recognized as constituted of well-developed and typical tissues and organs particularly situated at curiously foreign situations; *e.g.*, tooth, skin, nails, and hair, often found in a dermoid in the ovary.

The peculiar features of the teratomata are that the assortment of the different physiological tissues is of a varied nature, and the tissue elements are **not atypical** as in malignant neoplasms, but some of them are advanced developmentally which plainly resemble one or more of the fully developed tissues of the mature organism. Tissues and organs, namely, skin, nails, hair, mamma, nipples, tooth, bone, muscle, and even parts of organs, viscera, nerve, or as a matter of fact recognizable embryonal or fœtal parts are all detected in a Teratoma; *e.g.*, Ovarian Dermoids, which is a typical example of such growths.

Under the description of Teratomata therefore we shall describe the following varieties of tumours as a matter of convenience.

- A. Teratomas of totipotential cells.
- B. Blastomata of pluripotential cells.
- C. Teratoids, or Blastomata of Teratoid nature, of unipotential cells.

A. Tera-  
tomata  
proper.

## A. TERATOMATA.

**TERATOMA**, is an autonomous neoplasm growing as a result of continued but incomplete and imperfect development within one individual of another individual of the same species. The origin of the latter may be due to ; (i) excessive or reduplicated growth of the germinal area with the production of an imperfect second fœtus, as it perhaps happens in sacrococcygeal, *vide* photo plate No. LIII, and cranial tumours ; (ii) dislocated blastomeres. At a very early stage of development the cells formed by the segmentation of the ovum are called blastomeres. Some of these blastomeres may become displaced and arrested in their growth, which may at any time light up to activity and produce cells and tissues of all three primitive blastoderms ; (iii) process of perthenogenesis, or development of an ovum or spermatozoon without fertilization or fecundation. All these above suggestions are mere theories or speculations.

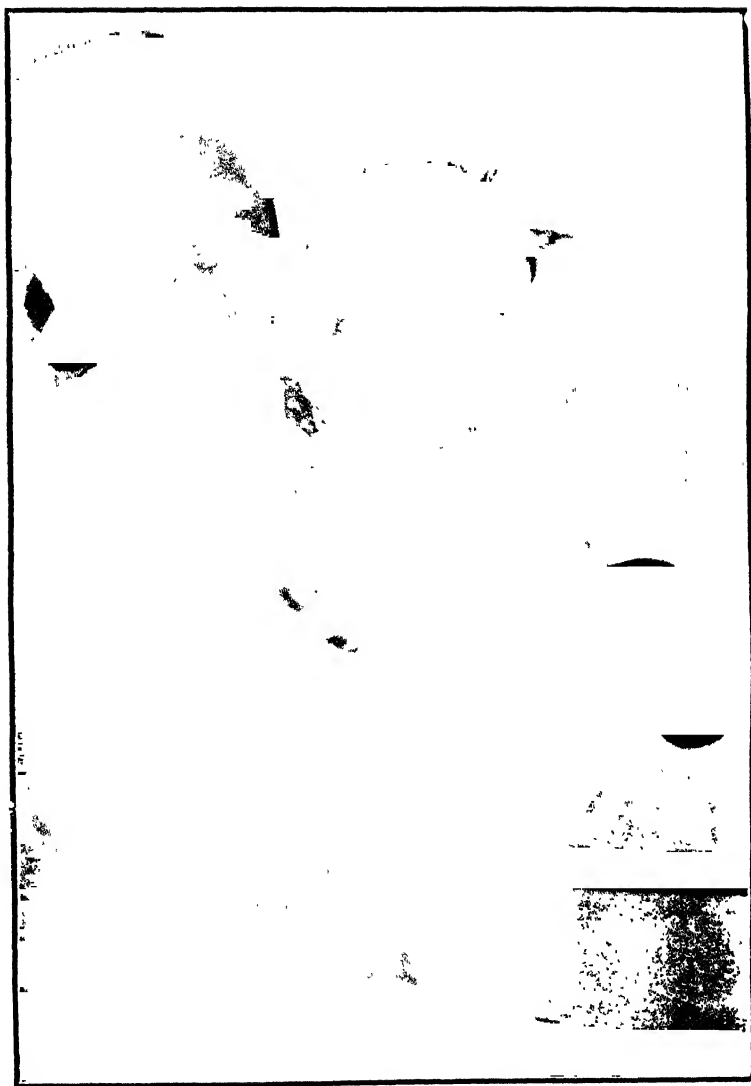
Important  
Surgical  
points.

Teratomata  
usually  
develop  
in the  
generative  
glands.

Majority of  
testicular  
tumours are  
teratomata.

Metastases  
may be uni-  
potential  
blastoma,  
or a toti-  
potential  
teratoma.

But the most important surgical point is that most of the important and typical as well as commonest examples of Teratomata are seen in the genital organs, *e.g.*, the **ovary** and the **testicle**; and that extragenital Teratomata are rare and of little clinical importance so far as practical surgery is concerned. Moreover the majority of the testicular tumours now-a-days are believed to be Teratomata. It is very important to remember that usually a teratoma of the testicle remains dormant for some time deceiving us to believe its nature as apparently non-malignant. As it may not show its malignant nature from the very beginning it is wise practically to regard every Teratoma of the testicle as **potentially** malignant. General dissemination may therefore be delayed, or may occur early. It is not necessarily that the secondary growths should be represented by cells of all the trilinear blastoderms. They may be limited to the nature of unipotential blastoma, or may faithfully reproduce a totipotential teratoma.



SACRO-COCYGEAL TERATOMA.



Teratomata may be **solid** or **cystic**. The solid types are more important to a general surgeon, as Testicular Teratomata are *practically always* solid; and a cystic variety of teratoma of the testicle of the nature of a **dermoid** is *extremely* rare. To a gynæcologist on the other hand the cystic type of teratomata is more important as the majority of the ovarian tumours are of cystic type called Ovarian Dermoids. Solid ovarian tumour of teratomatous type is on the other hand rare.

Solid type more important to a Surgeon.  
Cystic type concerns the gynæcologists.

It must be generally understood that the solid type of the Teratomata either of the testicle or of the ovary is a more anaplastic type, and therefore malignant in nature. This type tends to disseminate early although the metastases may consist of cells of one order, or of mixed type.

Solid type is a more malignant type in nature.

Teratomata are tumours of early adult life, but may be present even in quite young children. They are often bilateral.

Teratoma is a tumour of the young.

**TESTICULAR TERATOMATA**, although described as generally solid, present a variable picture. Innumerable tiny cysts of variable sizes are more or less present in all the tumours. That is to say some tumours are more solid in consistence, exhibiting a few macroscopic cystic spaces, others at the other end of the pole may present many small degeneration cysts, justifying its old name **fibro-cystic disease** by which they were formerly known and described. Sometimes the cysts are large enough to give it an appearance of a sea-sponge.

Testicular Teratomata.

Majority of testicular tumours are teratomata.

**MACROSCOPICALLY**,—a Teratoma in the testicle consists of a firm rounded tumour of dense tissue, which on section presents a variable picture exhibiting innumerable solid and cystic spaces, resembling a sponge as described above. Hæmorrhage, œdema and other degenerative changes and disproportionate dilatation of one or more of the cystic spaces may give rise to various appearances of the general structure on the surface of the section, in which hairs, cartilage tissue, glandular tissue, muscle tissue may all be detected.

Macroscopic appearance.

**MICROSCOPICALLY**,—the structure of the tumour consists of a collection of tubules lined by columnar epithelium and surrounded or separated by various other tissues such as bundles of smooth muscle fibre, the whole lying in a stroma composed of loose fibrous tissue. At other different areas islands of squamous epithelium, hairs and masses of cartilage may be seen; or as a matter of fact by careful examination derivatives of all three of the primary layers of the blastoderm can be found; and therefore any particular area of a section may be readily mistaken for an adenoma, or a chondroma, or a myoma. In some areas again it may be distinctly mistaken for a carcinoma as many of the epithelial cells may remain embryonic or undifferentiated. *Vide* photo plate No. XLVIII.

For clinical features, diagnosis and treatment, the reader is referred to regional surgery.

Ovarian  
Teratomata.

**OVARIAN TERATOMATA**, occurs under similar conditions as the testicular type. But as stated above, the cystic type is more common than the solid variety. The solid Ovarian Teratoma is a very rare tumour but very malignant and the cells composing them are of more anaplastic type, and therefore disseminate early. Otherwise it exhibits the same conglomeration of tissues as the cystic variety described below.

Cystic type  
is called  
**dermoid**,  
very  
common  
type of  
teratoma.

**DERMOIDS**, or the cystic type of Ovarian Teratoma may be unilocular, or multilocular. Sometimes it attains a considerable size, and in malarious countries may often be mistaken for ascites. But in contrast to such a condition it is not so malignant. The tumour on section is found to contain pultaceous or caseous material and composed of cystic chamber or chambers, which may contain hair, tooth, and other tissues and organs lying free or in bundles inside them. Sometimes nodular projections may be observed in the inner surface of the wall of the cyst where teeth may be inserted or hair attached, representing the anterior extremity of the imperfect embryo.





PAROTID TUMOUR (CHILD).

Microscopically the cyst wall is lined by squamous epithelium underneath which hair, sweat and sebaceous glands representing epidermal tissues are seen. Cartilage, bone, muscle, glandular elements, nervous elements are very easily detected round about, sometimes arranged into structures resembling an organ such as the trachea, or the intestine. This is particularly noticed at the area of a nodular protuberance representing the head.

For clinical features, diagnosis and treatment the reader is referred to regional part of the Surgery.

This finishes the description of the common forms of Teratomata. The other two varieties of tumours described below in this group cannot be properly called Teratomata; but there are grounds, although full of doubts, in their favour to claim their relationship, however flimsy, with Teratoma. For various difficulties and stronger grounds they could not be included under the description of Blastomata constituted of unipotential elements.

## B. TERATO-BLASTOMATA OR BLASTOCYTOMATA.

B. Terato-  
Blastomata

**TERATO-BLASTOMATA**—are tumours derived from **pluripotential** cells of the individual in which they grow. That is to say they are **autochthonous** in nature but they are capable of producing many kinds of cells; instead of one as produced by unipotential blastomata.

All "**mixed**" tumours so called may be included in this group. "Mixed" tumours.

"Mixed" tumours occur in several situations, and some of them cannot be differentiated from true teratomata, as both these types are shared by some paired organ in a characteristic way; *viz.*, teratomata in the testicle and ovary, and terato-blastomata in the kidneys, salivary glands, eyes, *cruræ cerebri*, etc. These are the tumours also described as **blastocytomata** by some authorities. These are also called blastocytomata.

These are the tumours of indifferent cells.

on the supposition of their being derived from **indifferent cells** or **Blastomeres**, that is to say capable of producing both epithelial and mesoblastic elements.

Terato-blastomata in the majority of instances occur in the salivary glands and the kidneys.

Renal terato-blastomata.

**RENAL TERATOBlastomata** occur in the majority of the instances during infancy especially before the end of the second year. These tumours attain a very large size, they are more or less firm in consistence, and of a whitish appearance.

Adeno-Sarcoma.

The microscopical appearance of the main mass exhibits rounded or oval cells of distinctly sarcomatous type intimately mixed with tubules lined by columnar cells lying in a mucoid or œdematous connective tissue matrix. It is for this peculiar structure these tumours have been described under many different names with different significance; *e.g.*, Carcinoma-sarcomatodes, Adeno-Sarcoma, Rhabdo-myosarcoma, Sarcoma of infancy, etc. In some instances smooth and striated muscles, and irregular islands of cartilage cells can be made out. But the most important point is their absence of exhibiting any tendency to arrange themselves in any orderly grouping of the different elementary constituents such as is seen in the true types of Teratoma.

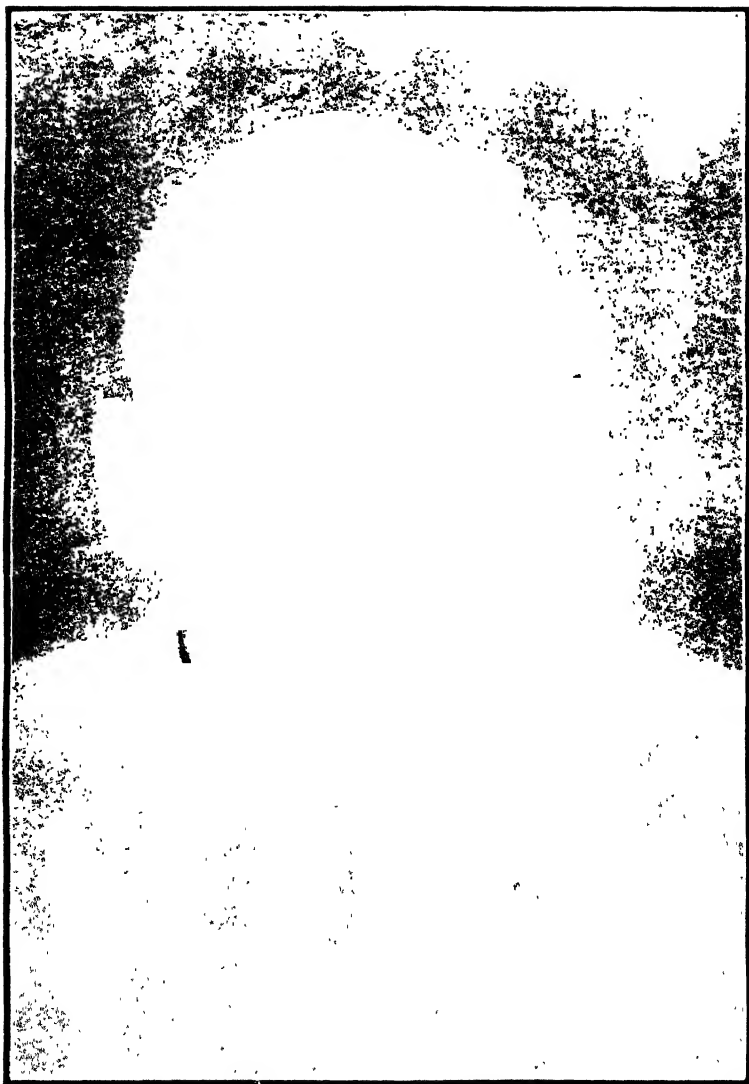
Wilms's Tumour.

These kidney tumours are now-a-days described as Wilms's tumour, after Wilms who traced their origin to a mesoblastic rest. It is the primitive segment of the body which gives rise to the different connective tissue of the body wall, bone, cartilage, striated muscle, etc. And it is from this primitive segment the kidney anlage also arises during development. Any arrest of a displaced group of the above connective tissue cells in the substance of the kidney may be the source of acting as the starting point of the tumour.

Very highly malignant.

These tumours are highly malignant, metastases occur in all instances, and they are locally very infiltrating.





PAROTID TUMOUR (ADULT).

**MIXED TUMOUR IN THE SALIVARY GLANDS.—**

Mixed  
tumour in  
the salivary  
glands.

Although tumours occur in all the salivary glands they are far more commonly seen in the **parotid**, and tumours of "mixed" character in the parotid are usually described as **parotid tumours**.

**PAROTID TUMOURS**—are usually composed of a mixture of various kinds of cells, the origin of which is still an unsettled question. But in contrast to the previous type of "Mixed-celled" tumour in the kidney "mixed" tumour in the parotid remains remarkably non-malignant for a number of years; at any time during which it may light up to a malignant character excited by some real or apparent cause. In the form of a non-malignant tumour also it is one of the simple tumours which is very commonly met with. *Vide* photo-plate No. LIV.

Parotid  
tumour  
remains  
usually non-  
malignant  
for a long  
time.

**MACROSCOPICALLY**, it appears as a slow-growing, painless, nodular, or lobulated swelling of fairly hard consistence, generally commencing at the level of the duct or the lobe of the ear, often attaining a size as large as a cricket ball. *Vide* photo-plate No. LV. The skin over the swelling remains movable throughout, showing no tendency to malignancy or lymphatic gland invasion for a considerable number of years after which as a rule the tumour becomes malignant. On section a fairly firm semitranslucent surface mottled with whitish area of denser growth is exhibited.

**MICROSCOPICALLY**—it consists of a heterogenous mass of cells resembling more or less of endothelial type, for which reason it used to be called an endothelioma. But various types of cells, some cubical, others polygonal or flattened, occur in solid masses, or form narrow branching strands frequently lining lumina or spaces, in a matrix made of fibrous, cartilaginous, nævoid, lymphoid, and myxomatous tissues. At various areas extreme mucoid or colloid degeneration is seen which is a distinctive feature of this tumour. In old standing cases

Parotid  
tumour  
is a real  
teratoma.

Clinical  
features.

again at certain areas and at times perfectly formed squamous epithelium with prickle cells and keratinization, or sarcomatous elements are often present. Isolated cells resembling cartilage cells may be formed in the condensed myxomatous stroma, and in some instances true cartilage may be present; or even in some tumours bone tissue may be made out. The parotid tumour is sometimes described as Mixed-celled Chondro-adenoma. It is for this mixture of cells of pluripotential nature parotid tumours possibly belong to real **teratoma** group.

CLINICALLY—the tumour may appear at any age, usually starts about the age of twenty-five or thirty, and may remain non-malignant without giving any trouble excepting the size for a considerable number of years, even twenty or twenty-five; when it may start a life of malignant character, especially if excited by some misadventure in the shape of irritation or quack-doctoring. (*Vide* photo-plate No. LVI). With the growth of the tumour mastication is mechanically interfered with, which in some instances may get jammed in between the sterno-mastoid muscle and the angle of the jaw. Extensions and processes may encroach upon the masseter and down to the styloid process. But the most interesting clinical features are, that it does not adhere to the skin, does not involve the facial nerve, and does not infiltrate the gland unless and until it becomes malignant. Otherwise it presses the glandular tissue, the nerves, and the skin, and other neighbouring structures aside; and it appears to have and to retain a *definite capsule*, helping our operation comparatively easy during its extirpation. On palpation it may present a hard or soft consistence of variable degree.

Malignant  
transfor-  
mation.

It is not possible to predict whether or not, or at what time, the tumour will turn malignant. When malignant transformation takes place the signs of malignancy appear rapidly by exhibiting the infiltrating natures of a malignant tumour. The skin overlying it becomes



PAROTID TUMOUR (OLD)



fixed, the tumour becomes hard and painful, the mass soon gets fixed to the surrounding neighbouring tissues, and presses upon the vessels and nerves; when signs and symptoms involving the facial nerve appear more pressing, causing facial paralysis. The lymphatic glands are soon infiltrated and enlarged, and ulceration over the glands and even fungation over the tumour may soon occur. Metastases may take place even at distant sites, and cachexia soon sets in.

**DIAGNOSIS**—is easy as it is probable that the various forms of longstanding tumours in the parotid described as fibroma, adenoma, etc., are really of the nature of "Mixed" type.

**TREATMENT**—consists in complete extirpation, care being taken to leave no process or lobulation behind; since isolated lobules may escape notice at the operation, and result in a recurrence of the condition. It is always wise to remove the tumour before it shows any sign of malignancy. When signs of malignancy becomes apparent extensive operation including removal of the angle of the jaw and tying of the carotid may be required; for the details of which, and radium and other methods of treatment in inoperable cases, the reader is referred to the regional part of the Surgery.

### C. TERATOID-BLASTOMA OR CHORION-EPITHELIOMA.

C. Tera-  
toids.  
Chorion-  
epithelioma.

This is a class by itself having only one type of tumour. It differs from the former in being **heterochthonous** in origin; that is to say the tumour grows in the individual bearing a tumour but produced by certain cells of unipotential nature from another individual. There is only one member in this group, and that is **chorion-epithelioma**. This is a blastoma in the sense because its elements are unipotential in nature. It is teratomatous in the sense that it is derived from another

individual. It is not a proper Teratoma because it is not composed of cells derived from all the three trilaminar blastoderm, but only the epithelial cells. Whereas the "Mixed"-celled tumours are blastomatoid teratoma, this form is teratoid blastoma; and being composed of cells of unipotential nature and not tissues, are really **cytoma**; and not an organ tumour as the teratomata are. And thus it is **singled** out from all other known tumours. It is the type of tumours which are produced by inoculation on animals by transplanting cells from human tumours for experimental purposes in the laboratory; the tumour cells supply the seed and the elements to grow, and the host maintains them.

We have already described the peculiar features of this type of tumour with reference to its origin. It is by itself a very rare tumour, and although in the majority of the instances it grows in the uterus of a pregnant woman after abortion especially in a multipara from cells derived from the foetus, it is also seen in non-pregnant uterus, and even in the testicle of the male sex. These latter features turn the question of its origin and nature highly debatable and controversial.

In a large proportion of cases the development of Chorion-epithelioma is preceded by a hydatid mole occurring either in the same pregnancy or in the one preceding the recent one.

**SITE.**—The tumour may be situated anywhere in the genital tract; namely, the uterus, the ovary, the Fallopian tube and the vagina.

The tumour is called Chorion-epithelioma as the neoblastic cells are derived from the cells of the chorionic villi of the fetus. Clinically three varieties of this tumour are met with; *viz.* :—

**Varieties.**

(1) Uterine.

(1) Uterine types.

(a) Chorio-carcinoma.

(b) Syncytioma.

(2) Testicular type.

(2) Testicular.

(3) Extra-genital type.

(3) Extra-genital.

(1) **THE UTERINE TYPE** is the one usually seen, and is derived from the foetal chorionic villi.

(1) The Uterine type.

This chorionic villi are normally covered by two layers of ectodermal cells, the function of which is to invade the maternal blood sinuses; but the villi are destroyed at the later stage of pregnancy. These layers are composed of: (a) the Langhan's cells, or the inner layer of the trophoblast covering the villus. (b) The Syncytial cells, or the outer layer of the trophoblast covering the villus.

Both these layers are responsible to give rise to cancer of the most virulent type ever known in women, as both are very **invasive** in function. The types met with are:—

(a) **Chorio-carcinoma.** This is derived from the Langhan's cells. These cells are cubical or polygonal with relatively large nuclei in a very clear cytoplasm arranged in several layers. Tumour arising from these cells is usually preceded by hydatid mole in the preceding pregnancy, or abortion in the same pregnancy. This type is very malignant and metastases are always seen.

(a) Chorio-carcinoma  
Very malignant.

(b) **Syncytioma** consists of groups and islands of syncytial cells, in which the bulk of the mass is made of necrotic tissue and blood clot. The cells are often giant cells in type. But the peculiar feature of the type is its markedly regressive tendencies, and its absence of producing metastases.

(b) Syncytioma.  
Metastases absent.

As there are *intermediate* varieties in between the two types it is clinically difficult to distinguish them.

**CLINICALLY**, in a case with a history of pregnancy followed by abortion, especially in a multipara, a growth may originate at the placental site which increases in the form of a polypoid tumour. The mass is

Clinical features.

soft, hæmorrhagic and maroon coloured. The uterus becomes enlarged in size due to the enlargement of the tumour which exhibits a curious tendency to the formation of implantation growths on the lower part of the uterine wall, sometimes extending on the wall of the vagina. On the other side it invades the muscle and appears on the peritoneal surface of the uterus.

Dissemination takes place very rapidly by the blood stream as the cells are in direct relationship with the maternal sinuses, and the metastases are found in the lung and liver lodged by the arrests of the emboli.

Some cases of Chorion-epithelioma is found to disappear spontaneously followed by complete recovery. Others again are seen to remain non-malignant. In some very malignant instances even the metastases also disappear with the primary tumour when spontaneous recovery takes place.

Treatment.

**TREATMENT**,—consists in complete pan-hysterec-tomy, for the details of which the reader is referred to the regional part of the Surgery.

(2) Testi-  
cular  
Chorion-  
epithelioma.

## (2) TESTICULAR CHORION-EPITHELIOMA.

Testicular chorion-epithelioma is another manifesta-tion of chorion-epithelioma which exhibits still more interesting features.

Instead of growing from foetal tissue as occurs in uterine chorion-epithelioma it develops in a **teratoma** of the testicle. It arises by the formation of chorionic tissue from the embryonic rudiments of a teratoma, which in turn gives rise to a blastomic growth which exhibits in every respect the character of a typical Chorion-epithelium of the uterus. Here the teratoma takes the place of the foetus and behaves exactly as it does in the uterus.

Testicular chorion-epithelioma is intensely malignant. Dissemination takes place *via* the veins, and secondary

deposits soon start in the lung, liver, long bones and brain.

Treatment consists in extirpation and abdominal orchidectomy with the lumbar glands "enbloc;" for the details of which the reader is referred to the regional part of the Surgery.

### (3) EXTRA-GENITAL CHORION-EPITHELIOMA.

(3) Extra-genital Chorion-epithelioma.

Extra-genital chorion-epithelioma may arise as a primary tumour, when it usually occurs in the thorax; or as secondary tumours developing from metastases arising from a primary growth in the uterus or testicle.

These are of little interest to a practical operating surgeon.

## SUMMARY.

### II. MESOBLASTIC CELL TUMOURS.

#### SARCOMATA.

Two principal parts of Sarcoma, *viz.* :—

- (i) Parenchyma or cells.
- (ii) Stroma of Fibrous Tissues.

Stroma has three parts.

Sarcomata are classified according to the variations of their parenchyma cells. The growth and Metastasis.

Sarcomata of undifferentiated cells.

- (i) Round-celled Sarcomata.
- (ii) Spindle-celled Sarcomata.

Oat-celled Sarcomata. Alveolar spindle-celled Sarcoma. Alveolar Sarcoma.

Organized Sarcomata :—

- (1) Lympho-Sarcoma.
- (2) Lipo-Sarcoma.
- (3) Fibro-Sarcoma.
- (4) Osteo-Sarcoma.
- (5) Chondro-Sarcoma.
- (6) Leiomyo-Sarcoma.
- (7) Rhabdo-myo-sarcoma.
- (8) Glio-sarcoma.
- (9) Neuro-sarcoma.
- (10) Myxo-sarcoma.

### III. PIGMENTED TUMOURS.

Nævus cells. A Nævus may be pigmented or non-pigmented. Histology of the Nævus cells. Two main varieties. Non-malignant Pigmented Tumours. The Nævus or Mole. Two forms. (a) Infantile Form. Important surgical point. (b) Adult Form.

Clinical Features. **Melanotic whitlow.** Treatment. Important Surgical Point. **Melanomata** or malignant pigmented tumours. Melanomata. Melanosis. Melanuria.

### IV. HYPERNEPHROMATA.

Boyd's Law. Cut Pomegranate. "The Golden Tumour." Autochthonous Blastomata. Heterochthonous Blastoma. Chorion-Epithelioma.

### V. TERATOMATA, TERATOIDS.

#### A. Teratomata Proper.

Important surgical points. Teratomata usually develop in the generative glands. Metastases may be unipotential blastoma, or a totipotential teratoma. Solid type more important to a Surgeon. Cystic type concerns the Gynæcologists. Solid type is a tumour of the young. Testicular Teratomata. **Majority of testicular tumours are teratomata.** Macroscopic appearance, Ovarian Teratomata. Solid type very rare. Cystic type is called **dermoid** very common type of Teratoma.

#### B. Terato-Blastomata. **Blastocytomata.**—

"**Mixed**" Tumours. These are called Blastocytomata. These are the tumours of Indifferent Cells. Renal Teratoblastomata. Adeno-Sarcoma. Carcinoma-sarcomatodes. **Wilms's Tumour.** Parotid Tumours remain usually non-malignant for a long time. **Parotid Tumour** is a real Teratoma.

#### C. Teratoids.

Chorion-epithelioma. Varieties.

##### (1) Uterine Type.

(a) Chorio-carcinoma. Very malignant.

(b) Syncytioma.

##### (2) Testicular Chorion-epithelioma.

##### (3) Extragenital Chorion Epithelioma.

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## CHAPTER VI.

### THE DIAGNOSIS OF SWELLINGS AND GROWTHS.

We have arrived at a stage of our learning where we find our knowledge acquired thus far is taxed by various different kinds of **swellings** we come across at the bed side in our hospital practice.

The diagnosis of swellings and growths

A **SWELLING** may be caused by a lesion starting in the form of an **abscess**, or a **cyst**, which we have studied before; or by any of the varied kinds of **neoplasms** we have just studied in this volume, or any displacement of some hard or soft tissues or organs or deformities caused by trauma or malformation.

A list of swellings.

A **SWELLING** may be constituted of any of the lesions enumerated below, *viz.*:—

- (i) An **abscess**, or exudation in the **tissues**.
- (ii) A hypertrophied **lymphatic gland**.
- (iii) An aneurysm or a dilated **artery**.
- (iv) An inflamed **vein**.
- (v) A hypertrophied or inflamed or fractured **bone**.
- (vi) Inflamed or dislocated **joint**.
- (vii) An inflamed or hypertrophied **nerve**.
- (viii) A herniated or displaced **organ**, or **viscus**.
- (ix) A **cyst**.
- (x) A **neoplasm**.
- (xi) A **deformity** or **malformation**.

(xii) A combination or a mixture of one, two or more of the above conditions.

At the time of studying the elementary surgery of an acute abscess we conceive the idea that by detecting only its four cardinal symptoms, *viz.*, **swelling**, **redness**, **pain** and **heat**, it is easy to diagnose an abscess absolutely definitely. But on finding the absence of **pain**, **redness** and **heat** in a chronic abscess our confidence in our know-

ledge of diagnosing the nature and the contents of a swelling becomes a little shaken. Yet while we depend on our knowledge of determining the presence of pus by detecting the **cystic** condition of such a swelling as a sure sign of diagnosing an abscess of chronic nature, with our progress still further on, our spirit of such a hasty diagnosis becomes damped on studying the different natures and contents of all kinds of **cysts**; as we find **cysts** contain clear fluid and not pus. After studying **neoplasms**, we suspect our knowledge hopelessly deficient even in detecting and diagnosing the nature and constituents of a chronic suppurative **swelling**, by our mere crude and hasty clinical methods. Diagnosis of a **swelling** therefore may be as easy as it may be difficult; as the **facts** upon which the diagnosis of its nature rests vary in different cases, as also according to different clinical stages and circumstances, of the same case. If we are methodical in our examination, careful in our observation, logical in our conclusion, cautious in our judgment, and watch our steps from the evil practice of jumping into a hasty conclusion at every stage of our incomplete examinations, deferring it till the very last, we may be able to solve most of the cases satisfactorily.

Methods of  
diagnosis  
of swell-  
ings.

I. Determine the  
(1) Site.  
(2) Position of the  
patient.

THE METHODS usually employed are as follows:—

I. First determine the actual: (1) **site**; and (2) **position**. At what **site**, on what particular member of the body, limb, or trunk or head, and where is the swelling situated should be the first question to be solved. In what **position** of the patient the tumour prominently exhibits itself; that is to say in what anatomical **structure** the Swelling is mainly **situated**, and how is it modified or affected when the patient is asked to change his **posture** and his **position**, such as standing, sitting, or lying, becomes the main question to be solved next.

(1) About the **site**; the first question to be solved, is the swelling situated:—

(i) On the line, or axis of a long **bone**?

- (ii) On the line of a **vessel**?
- (iii) In a particularly known **lymphatic gland**?
- (iv) In any septa or compartment occupied by a group of many **muscles**; or in the belly or body of a particular muscle?
- (v) On, or in a **bursa**?
- (vi) On, or complicated with any known **nerve**?
- (vii) Inside, or outside a **joint** associated directly, or indirectly with its movements?
- (viii) On, or implicated with a known **secreting** organ, or a gland, such as the liver, or the lung, bladder, testicle epididymis, ovary, kidney, etc., or any known anatomical organ misplaced or displaced?
- (ix) On, or in connection with, a **serous sac**, e.g., the pleura, meninges, spinal canal, peritoneum, scrotal sac, etc.; that is to say in anyway connected with any of the body cavities, viz., cranial, thoracic, or abdominal cavity?
- (x) Does the swelling appear to be on the skin only, that is if it is only superficial, or is it deep? Is the swelling visible at a glance apparently or obviously? All these factors should be carefully watched and recorded to weigh evidence, but they should not influence us to form an opinion at this stage.

Varices, reducible tumours, ascites, ovarian tumours, etc., are greatly affected by the different positions of the patient. Their detection requires certain manipulations which we shall describe under **palpation**.

II. Notice the **physical characters** of the Swelling. In conducting the examination of the physical characters of a substance we apply our **senses** to determine its physical existence. We can dream or imagine a phantom but we must perceive and conceive a substance. To do that at the first sight or touch it is safer to avoid imagination, and realize what is real by actual perception. In scientific observations one should avoid to be a day dreamer as the Indian mind is prone to be. One should have the eye, ear, touch, smell, and taste of a

II. Notice the physical characters

western materialist. In the present circumstance let us examine: (i) What we **see** with our eyes? (ii) What we **feel** by our sensation of touch? (iii) What we **hear**? (iv) Do we **smell** anything or get the smell of any discharge? In other words, we should try to elicit the signs and symptoms by **inspection, palpation, percussion** and **auscultation**, and corroborate our observations by other scientific examinations consisting of pathological, bacteriological, radiological, chemical, and other physical examinations; and hearing the **history** as well as watching the course subsequently.

I. By inspection.

## I. INSPECTION.

By **inspection** we may notice the following points in a Swelling, *viz.* :—

(i) Is it an actual swelling.

(i) **Is it an actual swelling?** By which we mean whether it is something of the nature of a protuberance, raised beyond the level of its neighbouring structures, out of proportion of physiological standard. A baby may be so overfed that his cheeks would appear to be very fatty and plump, but it does not appear to be physiologically out of proportion. A mother will be shocked to hear such a bonny cheek described as swollen. Similarly we do not describe a young nursing mother's hypertrophied mamma as swollen unless at any particular area it appears to be raised up beyond the actual surrounding contour although one mamma may become physiologically more full than the other. We must in such a case compare the other mamma to see if there is any actual inconsistency in size or contour. So also we do not describe a villous or warty proliferation as a Swelling.

(ii) Single or multiple?

(ii) **Is the Swelling multiple or single?** **Isolated** single swellings at different limbs of the body may occur in acute or chronic inflammatory condition of many glands, which may be general or specific multiple abscesses, or disseminated malignant growths. Many swellings in the same limb or a particular area of a

limb are usually nodular growths which may be due to some localized extensions of **granulomatous** conditions. A single swelling may be an abscess or a tumour or a cyst. This much we may say at this stage of our examination that the lesion under examination is one of a **single swelling**, and nothing more.

(iii) It may be a Swelling, but while inspecting, it is safer to recall atonce our knowledge of anatomy and physiology to determine if it is:—

A **deformity**,—and if it be so, is it a (1) **malformation**, or (2) a **misplaced organ**? *e.g.*, an **undescended** testicle at the groin, if it becomes painful will atonce mislead us to a wrong track unless we examine the scrotum beforehand to be sure if the patient has got both the testicles in his scrotal bag. A young girl may have an ovary congenitally herniated in the labia. A deformed or dislocated joint, or a fractured limb, congenital or acquired, or a mammary gland in the male, must be sought for and eliminated, otherwise there is every chance of such a condition being overlooked.

(iv) The **shape** of a swelling is the next point to be observed. What **form** has it assumed? Gas or fluid or semisolid substances pumped into a soft and yielding bag, or cavity, will distend and exert a uniform ballooning or hydrostatic tension and pressure all round, and therefore the form or shape of such a cavity or bag or whatever it may be, is expected to be, (1) **uniform** in contour all round. But the actual fact is, that in the human body even with contents of such consistence a swelling may become, (2) **irregular** in contour; for the simple reason that the wall constituting the sac is usually not of the same strength all round, and it may give way at a certain spot where a protruding portion of it may give rise to a lobulated or pedunculated outgrowth. This may also be due to excessive action of the bacteria at a certain area, or the physiological vulnerability of certain tissues, or other mechanical

(iii) Or a Deformity.

(1) Malformation or  
(2) Misplaced organ.

(iv) Shape.

(1) Uniform.

(2) Irregular shape.

Regular shape.	causes. A pathological Swelling of <b>regular</b> shape may have the <b>form of a normal physiological structure</b> of the body, and so demonstrate its relation with such a particular structure. We may be able to spot out a hypertrophied testicle or a kidney by the general shape of the swelling, and thus diagnose the swelling to be in relation to those organs, by the physiological shape and form the pathological swelling may have adopted.
(a) Physiological form.	A <b>globular</b> shape of a swelling indicates the uniform yielding of tissues, and its expansion subjects the tissues to a uniform pressure of the growth, or all-round equal proliferations in a rapidly growing neoplasm, <i>e.g.</i> , a <b>cyst</b> of any kind will assume a globular out-line. A <b>blood vessel</b> such as an artery due to weakening of its wall at a certain spot caused by some chronic disease of its wall may yield at a spot and allow the hydrostatic pressure of the cardiac circulation to forcibly pump the blood out of the course; yet gradually contributing to form a sac or a saccule to give room for the blood in that newly formed sac without bursting. These are called <b>aneurysms</b> to be described. Naturally they take a globular shape all round due to uniform distension. Similarly a <b>diseased joint</b> may likewise assume a globular shape when the fibrous structures and other connective tissues are softened, and are no longer able to maintain the normal outline of the part against the pressure of the synovial distension. Soft <b>medullary carcinoma</b> and <b>sarcoma</b> often assume a globular shape. Hard fibroma assumes a globular shape by uniform proliferation of tissues all round. Sebaceous cysts and dermoids may be distinguished from abscesses and fatty tumours by the former two conditions usually adopting <b>hemispherical</b> shape, and the latter assuming an outline of an <b>ovoid</b> nature. It must be remembered that an abscess never assumes the shape of a <b>full globe</b> or a sphere, but always adopts a <b>hemispherical</b> or <b>half-ovoid</b> or <b>half-dumb-bell</b> shape. A growth or a Swelling may also be <b>warty</b> , <b>villous</b> , <b>pedunculated</b> , <b>polypoid</b> or <b>lobulated</b> . In the chapters on neo-
(b) Pathological form.	
Globular.	
Cyst.	
Aneurysms.	
Diseased joint.	
Medullary Carcinoma and Sarcoma.	
Fibroma.	
Hemispherical.	
Ovoid.	

plasm we have seen how according to the nature of proliferation the clinical types of growths are classified. From the nature of the outline, or forms, or shapes, of these growths we can sometimes spot out a neoplasm from a granuloma, or inflammatory villous, or papillomatous, or nodular, or tuberos, proliferation of granulation tissue. A neoplasm or an inflammatory swelling may be **lobulated**, *e.g.*, a cold abscess becomes often lobulated. A flattened ovoid lobulation of a Lipoma may be very distinctive, but it may be mistaken for a cold abscess if one does not remember the actual characteristic features of a cold abscess and the more superficial nature of a lipoma; although sometimes diffuse lipomata may be a confusing growth. A herniated portion of omentum sometimes presents a lobulated appearance, although remaining inside a hernial sac; but when it remains interstitial in the abdominal parieties it resembles a cold abscess, so much so by the tension exerted by the surrounding muscular layers, that the real nature of the swelling may not be detected till at the last stage of the operation started with some other diagnosis. Usually a herniated omentum manifesting itself like a lobulated tumour may be easily determined by its granular and **loosely** lobulated feel. **Coarser** lobulation of tumours may be caused by the yielding of the surrounding tissues in certain directions, only of the least resistance. This condition occurs in Cyst-formation as in the hydrocele of the testicle cystic degenerations in the tumours of the mamma, or embryoma in the testicles associated with cyst-formation. The same condition may occur in some ganglia, of the small joints or in some forms of enchondroma. Lobulation of various forms, *e.g.*, dumb-bell shaped swellings, Indian club-shaped swelling, etc., often occur in psoas abscess. For details *vide* Cold Abscess, Volume II, Part I.

(3) Other-  
forms  
Warty,  
Villous,  
Pendun-  
culated,  
Polypoid,  
Lobulated.

Loosely  
lobulated.  
Coarser  
lobulation.

Omental  
hernia.  
Hydrocele.  
Cystic  
mammary  
Sarcoma.  
Cystic  
ganglia.  
Enchon-  
droma.  
Psoas  
abscess.

**RETRACTION** is a pathological feature, which dis-figures the shape of many physiological, or previously formed pathological tissues, and is usually seen in patho-

Retraction.

logical terminations of cicatrix. It is also a very characteristic feature of a scirrhus carcinoma.

Peau d'  
orange  
skin or  
Pig skin.

**PEAU D' ORANGE SKIN OR PIG SKIN.**—In some forms of carcinoma of the breast sometimes, although not commonly, an extensive permeation of the lymphatics immediately under the surface occurs. This permeation produces a general blockage of the lymphatics at the deeper strata followed by their obliteration by defensive fibrosis, resulting in an interference of the lymphatic drainage; with the result that a condition of solid lymphatic œdema of the skin over the breast is produced. The skin is swollen, but as the result of the fibrosis underneath, the points of the anchored hair follicles with the corresponding areas of the skin and the openings of the sebaceous glands are unable to rise. The latter become enlarged and present themselves all over the affected part of the skin in the form of shallow pits which make the surface look like the pig skin. The general area of the skin on the other hand becomes more and more cedematous, swollen, thick, and tough, resembling the peel or coat of an European orange. This retracted condition of the surface is clinically described as "**Peau d' orange skin**" or "**Pig skin**."

(v) Colour.

(v) **Colour.**—The colour of all tissues depends on the vascular conditions associated both with blood and lymph circulation. The colour of the surface of a swelling may be:—

Red.

**RED.**—But the term red signifies nothing in particular to us, as its equivalent vernacular term "**lal**" includes in its connotation all varieties of shades starting from light pink, rose, or deep pink colour, to deep red, scarlet, or purplish-red, or bluish-red hues. In surgical diagnosis the exact understanding of the shade gives an outer indication of many pathological conditions, very nearly helping us on most of the occasions to determine the diagnosis correctly. Arterial, venous, lymphatic conditions, hyperæmia, congestion, œdema, etc., are

very differently manifested in their characteristic manner according to different characteristic pathological conditions inside. For different diagnostic significance of redness *vide* Volume I, Part II.

**BRIGHT RED** colour resembling red oxide of mercury is the result of capillary dilation, as we come across in inflammatory hyperæmia, but the area of such redness in the latter condition is **uniform**; and it may be more or less **pink**. Such redness may be next due to extravasation of blood from vessels. This may be uniform or spotted. A gentle touch and a little pressure by the tip of the finger may elicit two conditions; *viz.*, the colour may persist, or it may be dispersed; in the latter case the spot where the finger is in touch while still pressing becomes blanched; to be immediately re-filled on withdrawing. This latter phase occurs especially in acute inflammatory condition, particularly so when such redness is associated with œdema. If the colour is not altered on pressure the discoloration is due to extravasation of blood; and the following two conditions may arise out of extravasation of blood, *viz.*, (a) such discoloration may consist of many **isolated spots** which are called **petechiæ**; or (b) it may be a diffuse discoloration of various shades fading away peripherally at different zones, having outlines which are usually circular but may be irregular. Such a red area is called a **contusion**, or it may be an **ecchymosis**. The disappearance of the red colour on pressure shows that the blood is still circulating inside the vessels, but its persistence exhibits the condition of its existence outside it, or in other words, **extravasated**.

Bright Red.

Pink.

Inflammation.

(a) Petechiæ.  
(b) Ecchymoses and Contusions.

Extravasation.

Dilatation of vessels larger than capillaries containing blood of a bright red colour when seen over **malignant tumours** indicates the involvement of the skin, and this is seen very characteristically in some forms of cancer of the breast.

**SCARLET RED.**—Some swellings exhibit a dark scarlet red colour. Such a hue may be produced by

Scarlet red colour.

**Venous congestion.** **venous congestion**, which is usually accompanied with cedema. **Nævus** sometimes appears dark scarlet red. **Hæmatoma.** **Hæmatoma** especially of more than two or three days' standing turns scarlet red. Some **degenerations in tumours** when they become hæmorrhagic, are exhibited by a scarlet red hue on the surface. **Spermatocele** often appears scarlet red on the surface especially when complicated with sarcomatous development inside it, but in such conditions the colour effect is not uniform and individual vessels can be seen with pale spaces between them.

**Purple.** **PURPLE** discolouration is a characteristic sign of extreme **venous congestion**. **Nævoid** conditions are usually manifested by purple tint, the hue varying from bright to dark red and then to purple in colour, according to the pathological varieties of the tumours, or such swellings of congenital origin.

**Brown.** **BROWN** or **green** colour is seen on the surface of a hæmatoma of a few days' standing.

**Green.**

**Black.** **BLACK** colour is seen on the skin of **melanotic** conditions. Gas gangrene is manifested by black colour on the surface. *Vide* Vol. I, Part I.

**PIGMENTED** and hairy moles become the seat of carcinoma or melanoma. Moles may enlarge to a non-malignant or a malignant tumour. Pigmentation is also met with as a result of degeneration of **Nævi**, or hæmatoma.

(vi) **Surface.**  
**Ulceration.**  
**Gas-Bubbles.**

(vi) The **surface** of the swelling should next be noted, to see if there is any **ulceration** on it. Any tumour of non-malignant or malignant character, may become complicated with ulcerative degeneration on the surface. Squamous-celled carcinoma exhibits a peculiar feature of its own. Ulcerative degeneration to malignancy may occur to non-malignant neoplasms. Inflammatory swellings may exhibit acute ulcerative condition. Infective ulcers, irritative ulcers, malignant ulcers, specific ulcers, have all their own special clinical features; *vide* Volume II, chapter on diagnosis of Ulcer.

GAS BUBBLES over œdematous swelling are signs of gas infection by *Bac. Welchii*.

After we have finished what we could elicit by mere inspection by our eyes, we may then proceed to use our hands to make use of our sense of **touch**, to perceive and conceive the nature and composition of the swelling as far as possible.

## II. PALPATION.

## II. Palpa- tion.

By **palpation** we examine and try to elicit what we feel. It is useless to use our fingers haphazardly to deduce a scientific observation. There is a method of conducting this process of examination. One should use at first only one finger, and exclude and eliminate certain facts by simply touching and gently pressing the swelling by the tip of that finger. He should then by his finger such as the index and the thumb gently try to pinch the surface of the skin up, and then lay all the four fingers flat, to feel and press the growth. He may then make use of all the fingers and the thumb of one hand only to hold or squeeze the swelling. Finally, one should apply both hands to elicit any fluctuation or actual mobility and adhesions. The examiner should then use his fingers for percussing the swelling to elicit the nature of the contents, and finish his examinations by mensurating the extent of the growth. Before a student starts his clinical studies it would be to his advantage to practise his fingers and hands so as to make a habit to work automatically *ad seriatim* as follows:—

In proceeding to examine by **palpation** we may first Method. use our index finger. By simply touching the swelling and very gently pressing it by the tip of the finger we may note two points, *viz.*; (i) Is the discoloration **dispersed**, and while withdrawing the finger does it **reappear** again at once? (ii) Does the swelling **dimple** and **pit** on pressure?

If the **discoloration is dispersed** by the pressure of the

tip of the finger, and it becomes blanched, the blood is existing intra-vascular, and it may be Inflammatory Hyperæmia or Venous Congestion. If the pressure does not disperse the blood it must be an extravasation, or the blood is extravascular, or it is a Hæmatoma. If the area becomes at once refilled as soon as the finger is withdrawn the swelling manifests the nature of capillary dilatation, and it is most probably inflammatory.

“Pitting on pressure.”

If the swelling **dimples** on pressure and disappears no sooner the finger is withdrawn the contents may be air or fat or fluid, and therefore it may be an inflated viscus as hernia, or a lipoma, or a cold abscess. If it pits on pressure which **remains** as a small simple dimple, and is **gradually filled up** again after the finger is withdrawn the phenomenon is described clinically as “**pitting on pressure.**” This is always due to fluid or gas **infiltrating** the **cellular** tissue. When such infiltration is produced by fluid it is called **œdema**. Œdema usually, but not always, obliterates the natural wrinkles of the part. Such swelling with œdema is usually associated with an acute or a subacute phase. When œdema gets a chance of remaining for sometime without being accompanied by retrogressive degenerations or necrosis, the fluid is hardened and organized. Acute or sub-acute œdema may be due to the following pathological conditions:—

- (i) Lymphatic obstruction.
- (ii) Acute inflammation.
- (iii) Venous congestion.
- (iv) Hydræmia.
- (v) Urinary infiltration, often described as extravasation of urine.
- (vi) Gas gangrene.

Œdema due to acute inflammation is accompanied with the four cardinal signs of inflammation, *viz.*, pain, heat, swelling and redness; and it is **localized**.

Œdema due to venous congestion is usually produced by some mischief in the return flow of the circulation, and it may either be due to something wrong in the heart

or vessels or blood; or on the other hand to some mechanical obstruction outside the vessel wall.

Œdema due to accumulation of serous fluid is painless, and is more or less due to some general cause such as cardiac, neurotic, renal, angeio-neurotic, etc., *vide* Volume II, Part II.

Œdema due to **extravasation** of urine is a rapidly progressive condition. It should at once be examined in its anatomical limits and extent and determined by other clinical history.

Œdema due to gas gangrene is characterized by the gas bubbles, and emphysematous feel.

Having thus elicited the above factors, and adjusting our observations to a certain extent, we next use our index and the thumb to try to pinch the skin of the surface up into a fold, and to observe if it is possible to lift the skin from the body of the swelling. This manipulation will give us an idea of any adhesion that may have occurred in the growth. We have seen that when deep seated malignant growths involve the skin the latter gets fixed with the growth and it cannot be pinched up. Regarding adhesions with other tissues we shall examine them later.

Palpation may then be done with all the fingers of the hand, being placed flat. By this means we may elicit the following points:—

- |  |   |
|--|---|
| (i) Is it <b>hot</b> or <b>cold</b> ?  | Palpation with all the fingers.                                   |
| (ii) Is it <b>tender</b> ?   |   |
| (iii) Is it <b>crepitant</b> ?   | Examine Temperature,  |
| (iv) Is there any <b>egg-shell, crackling?</b> or <b>fine dry crackling?</b> | Tenderness, Crepitation, Egg-shell Crackling, Fine dry crackling, |
| (v) Is it <b>emphysematous</b> ?   | Emphyse-  |
| (vi) Is it <b>gurgling</b> ?   | matous condition,   |
| (vii) Is there any <b>pulsation</b> ?  | Gurgling, Pulsation.  |

A **hot** swelling is inflammatory, a **cold** swelling may be a cold abscess, or a lipoma, or a cyst. **Tenderness** elicits inflammation of tissues or involvement of nerves, although it is usually due to inflammation. A **crepitant**

swelling if it is less dry and of a lower pitch is produced by some thin osseous or cartilaginous growth inside. When the crackling is very dry and high-pitched resembling something like the sensation produced by crushing an egg, it is described as "**egg-shell crackling**," and this is met with in Myeloid growths; and is caused by the yielding of the osseous proliferations inside it. This is a useful indication of the expansion of bony or cartilaginous tissues in the form of a thin plate growing inside a tumour, especially when it affects the articular end of a long bone. Similar sensation may be elicited in a few days old cephalhæmatoma where the pericranium yields under the finger. Sometimes dental cysts exhibit the same sensation if pressed. **Emphysematous** condition indicates the presence of gas or air bubble inside as happens in Gas-gangrene. **Fine dry crackling** sensation is elicited where the gas is contained in the connective tissue spaces in small quantities. **Gurgling** is a sign of having some viscus, *e.g.*, the gut inside a swelling, and it is met with in intestinal hernia especially during reduction. Its presence indicates the mixture of liquid with gas. **Pulsation** shows that the tumour is an aneurysm, or it is in relation with some artery, or the tumour is situated at its neighbourhood, transmitting the thrill of the cardiac impulse, indicating a special connection of the tumour with the **arterial** system in some way or other. It must be remembered that pulsation is observed where there is some obstruction to the passage of blood in its onward flow from the arterial system. A diffusion of blood in the subcutaneous loose tissue extravasated from a ruptured artery will produce no pulsation. Pulsation in a tumour may be due to: (a) direct communication with the heart, *e.g.*, aneurysm or aneurysmal varix; (b) to the presence of numerous small arteries in some sarcomatous tumours or bronchocele; (c) to direct contact of the tumour with an artery transmitting its pulsation to the surface. Abdominal neoplasms over aorta, popliteal glands, thyroid gland, growths on the clavicle are

Pulsation  
is due to  
what?

mistaken for aneurysms, for the pulsation they transmit from the big vessels lying underneath them. For the diagnosis of Pulsating Tumours *vide* Volume on Inflammation in Bone.

We may then hold the tumour with our fingers and thumb and try to lift it up from the body, attempt to see through it, and then attempt to squeeze the contents. By this we elicit three points, *viz.* :—

(i) Its **consistence**—if it is hard or solid, or soft, or doughy? Grasp the tumour and hold it up. Examine.

(ii) Its **translucency**?

(iii) Is it **reducible**? Can we empty the viscus or its contents?

A tumour may contain gas, or fluid or solid tissues and detritus, and the **consistence** may accordingly be soft or yielding or cystic or solid. Such consistence may not be uniform. One part may be cystic the other doughy, and another portion may be solid to the feel according to the nature of the contents. Not only a tumour may vary in consistence in different parts, but it may exhibit consistence of different nature at different stages and times. A soft cystic sac may appear inside the body of a hard tumour, or *vice versa*, a hard mass may grow inside a soft or doughy tumour. The soft sac inside a hard mass may be due to any of the three things, *viz.*, (a) suppuration or necrosis, (b) cyst-formation, (c) degeneration. Signs of inflammation may help us to suggest suppuration, but it is difficult to draw the distinction between cyst-formation and degeneration, as the softening may be due to cystic-degeneration. A cyst may exhibit a uniform tense globular collection due to uniform hydrostatic pressure all round. Conclusion based on such a factor may possibly be correct, but not likely. Softening may occur in tuberculous gland or secondary epithelioma in the glands; the former is a suppuration the latter is a degeneration. A gumma may encounter the same fate due to softening of the central core. Cystic hygroma, cystic sarcocele are usually associated with variation of con-

(i) Its consistence.

sistence. A hard growth in a soft mass indicates induration or sarcomatous proliferation of osteoid cells.

(ii) Translucency.

**Translucency** is tested by grasping the tumour, so that it is made tense and the skin tightly stretched over it, while a light is held close to it at the opposite side of the observer. Excepting a beam of light allowed to be reflected on it at a spot of the tumour the other surrounding rays are screened off. The observer will then be able to see the light passing **through** the body of the tumour, if the contents are translucent, in the form of a diffuse pinkish glow; especially if he looks through a wooden stethoscope or an improvised tube made of paper, placed firmly on the surface of the tumour. Translucency is produced by the collection of translucent fluid in the body, *e.g.*, synovial or serous. Sometimes by this method the position of the testicle and its size may be fairly guessed in a hydrocele.

Local translucency may be met with apparently on the surface of the swelling caused by a pointing abscess. A spina-bifida with a thin sac, sebaceous cyst, some nævi, dilated veins, etc., appear apparently translucent on the surface.

(iii) Reducibility.  
Squeezing.

While grasping a tumour we may try to gently squeeze it, to test if we can reduce its size, or **empty** its contents. A swelling is **actually** reduced in size when its contents could be emptied or pushed out of its sac or capsule; but it may also be **apparently** reduced and may not be completely emptied out. The contents may thus be made to pass when it can be successfully emptied, into; (a) one of the body cavities, (b) in another part of the sac or capsule situated inside a body cavity, or elsewhere, joined by a narrow tunnel, (c) in some instances into the vessels of the part. For instance, we can easily squeeze the contents of a reducible hernia from its new position inside, into the abdominal cavity, and if this occurs completely "en bloc" after manifesting a peculiar characteristic gurgling, the diagnosis may at once be established. Some forms of congenital hydrocele exhibit the same

feature. Tumour of the spermatic cord and chryptorchism may be similarly manœuvred. In all these instances the tumour empties into the abdominal cavity. A psoas abscess may have a dumb-bell shaped sac having one part inside the abdominal cavity and the other outside on the body surface. The outside one can sometimes be easily obliterated by pressure. Varix and some forms of aneurysm may be pressed gently to empty the contents of the swelling into the blood vessels according to the direction of their respective flow. In the appendicular regions of the body all reducible tumours contains fluid in the form of blood, serum, synovia or pus. In the axial region as the trunk or head the contents of the tumour may be solid or fluid, *e.g.*, hernia or meningocele or varicocele.

Squeezing is not the only clinical method employed to examine the variations of the size of a tumour. The other useful tests employed are; (a) **pressure** on the surface by the examiner; (b) the **effort** or **strain** exerted by the patient when asked to do so in a particular way directed; and (c) **position** the patient adopts. All these three manipulations aid us considerably in making a correct diagnosis.

Variation of size is also tested by,  
(a) Pressure,  
(b) Effort by the patient.  
(c) Position adopted by him.

When exerting pressure on a tumour, the *time* taken by the tumour to be reduced in size, or be emptied of its contents, and the *amount of resistance* offered by the consistence of the tumour sometimes enable us to judge the nature of the tumour, *e.g.*, tumours which on pressure "slips" in with a **sudden** motion are usually solid. Intestines when herniated out in a sac may be reduced by taxis, and this reduction when successful occurs quickly and is often accompanied by a gurgle. Semi-solid or cystic tumours, *e.g.*, cold abscess, hydrocele, etc., are **steadily** and **gradually** reduced according to the size of the aperture for reduction, and the resistance of other viscus inside the body cavity into which they are reduced. Varix are very quickly reduced by pressure, but it is dangerous to

apply this method to all aneurysms. Sometimes a fine thrill is detected by the finger during reduction showing that the contents of the tumour are fluid, but contain numerous small solid particles.

**Partial** reduction may occur when a reducible tumour, *e.g.*, an inguinal hernia is associated with an irreducible hydrocele. The tumour is only reduced to the extent of the amount of the gut or omentum it may contain leaving the hydrocele out.

The way the swelling is refilled, and the effect of pressure upon individual blood vessels at the efferent or afferent current may give valuable hint to correct diagnosis in some swellings. This is especially the case with aneurysms and varix.

**Effort** or **strain** exerted by the patient produces marked distension of the tumours which are in connection with the body cavities, the latter having some passive resistance of their own; or of the tumours when directly connected with the circulation of blood and lymph towards the heart. The impulse felt in a hernia during coughing or straining, or filling out of a hernia during such efforts is very characteristic. Any cystic tumour produced inside a body cavity but having an aperture to communicate with the exterior will produce the same kind of impulse on straining, *e.g.*, cold iliac abscess, psoas abscess, congenital hydrocele, hernia-cerebri, cranial or spinal meningocele, pneumocelc, etc. Increased tension inside a tumour produced by obstruction to the return circulation on straining, is seen in varicocele, and venous nævi, or in cranial tumours which get an extra supply of cerebro-spinal fluid by the pressure exerted on them. The beginner while attempting to feel an impulse should learn to distinguish between a true **impulse** and a **displacement**. Impulse is a filling out of a swelling and is more or less hydrostatic in nature, but displacement which is apt to be mistaken for impulse is a mere thrust forward of a solid tissue.

**Position** as adopted by the patient in the course of our examination aids to our diagnosis considerably. The variations of the size of a tumour associated with changes of position are effected either by gravity or by buoyancy of the tumour to some surrounding fluid, or the influence upon the swelling, due to the altered tension of muscles and fasciæ produced by the changes in the position of the limb. Varices are reduced when the limb is raised. Umbilical hernia sometimes disappears if the patient is laid down. Ovarian tumour can be diagnosed correctly by altering the position of the patient, *vide* Regional Surgery. Effusion into the bursa between the semi-membranosus and the inner head of the gastrocnemius appears like a prominent swelling behind the inner side of the knee when the knee is extended, being pressed out by the tension of the stretched muscles; but it disappears when the knee is flexed and the muscles are relaxed, the fluid in this case remains bulging the sac towards the space of the ham.

Having thus finished all our observations made from our examination by one hand, we proceed to use both the hands to elicit the following additional phenomena:—

Examina-  
tion by both  
hands.

(i) **Mobility** and its **range**.

During,—

- (1) Passive movement.
- (2) Active movement.

(ii) **Adhesions**.

(iii) **Fluctuation**.

- (a) Hydrostatic Tension.
- (b) Elasticity.
- (c) Compressibility.

(i) **Mobility**.—In the course of our examination when we try to elicit some clinical signs during the time of the patient's making an effort, or changing his position we may make use of another hand for further examination purposes. We may observe **mobility** of the tumour and its **range**; (a) during certain acts or active movements or

(i) Mobi-  
lity.

efforts made by the patient; (b) during some passive movements of the limb or part of the body concerned.

We may grasp the tumour by one hand first and determine its position; if it is in connection with any especial structure, *e.g.*, bursa, or any structure anatomically connected with a soft structure say the tunica vaginalis, or if it is a loose body in a joint, or something adherent in such a way with any bone or muscle or tendons as to be immovable, or moving 'en masse' from side to side in cross axis, or above downwards in longitudinal axis. In the case of some swellings, while the tumour is in our grasp we may ask the patient to make some efforts, *e.g.*, to try to swallow while we hold the goitre in our hand, to notice any change that may occur during deglutition; similarly we may try to feel any change of position of a swelling in the liver or enlargement of spleen during inspiration, and so on.

Hardening.  
Rigidity.

While thus grasping a tumour we may be mindful to feel any sensation of **hardening** or **rigidity** or contraction of a tumour under our hand. Such hardening shows a muscular movement, and proves that the tumour is most likely implicated with some muscular structures, *e.g.*, gastric tumours, intussusception, or the foetal movement of a gravid uterus. Mobility may be sometimes mistaken for fluctuation unless both the hands are properly employed, as described below under Fluctuation.

(ii) **Adhesions**.—We may then proceed to examine if any clue could be found for diagnosis from the relationship of the tumour with its neighbouring structure, and if we could be sure, so far as its **adhesions** are concerned to one or more tissues of the neighbourhood of the affected part. Adhesion is a sign of **great diagnostic value**. All tissues of the body, *e.g.*, epithelial, muscular, connective, osseous, or all systems, *e.g.*, nerves, vessels, glands, etc., in **normal** condition are more or less movable on one another. Surface skin is movable over our muscles, muscles are movable over each other, and all soft tissues are movable over bones. The only **exceptions** in this

physiological and normal anatomical conditions, where no gliding movement exists between tissues are the **scalp** and, mucous covering of the **hardpalate**, **gums**, the **teeth** and cutaneous coverings of the **palms** and **soles**.

Any Adhesion found between one structure with another means an abnormal condition. This pathological feature in acute condition is effected by round-celled infiltration, and proliferation of connective tissue cells, which with the period of chronicity or in some form of malignancy, is organized into fibrosis. These fibres make the structures adherent to each other. Any liquefying degeneration inside would result in corresponding fluctuating changes.

Diagnosis of Adhesions of a swelling should not be hurriedly decided upon, as the effect of such conclusion may be disastrous to our patient as the result of our adopting a wrong line of treatment. It should first be determined with what structure, or structures, adhesion has taken place; and during examination we should carefully elicit whether such adhesions are **real** or **apparent**. It would be unmistakably decided upon whether the swelling is actually adherent to any structure, *e.g.*, an exostosis may be growing out **primarily** from the bone tissue itself with which the tumour is adherent; or whether it is an apparent binding down of the tumour by some other tissues depriving the tumour of its independent movements, *e.g.*, a parosteal tumour; such adhesions being of **secondary** nature. Secondary adhesions are usually due to inflammatory or malignant infiltration. An acute and rapid all-round infiltration is a sign of suppuration which may be soon detected as fluctuation usually follows; but a sub-acute or chronic induration is a grave sign as it indicates malignancy. It is important to determine the particular tissue where adhesion has occurred.

Adhesions may take place to the **skin**. If we try to glide the skin over such a swelling, and if it is a primary adhesion of chronic nature arising from warts infiltrating

To the  
Skin.

into the deeper tissues it is a squamous-celled carcinoma. Adhesions to subcutaneous tissues may be tested by eliciting the mobility of any tumour under the skin and over the muscles. Otherwise a simple way to elicit the sign is to attempt to pinch the skin in a fold and try to lift it up as described before.

To the  
Muscle.

Adhesions to **muscles** may be elicited by asking the patient to put the affected muscles into action followed by contraction and relaxation. If on attempting to do so extreme pain is felt by him it is most likely associated with acute lesions. When adhesion takes place in the muscle fibres the tumour remains as mobile as the muscles concerned in both their longitudinal as well as transverse axes during relaxation of the affected muscles; but it becomes firmly fixed during their contraction.

To the  
Bone.

Adhesions to **bones** are easily determined by the fixity and the immovable nature of the tumour apart from the bone.

To the  
Gland.

Adhesions to **gland** are best elicited by grasping the gland, *e.g.*, the mammary gland, in one hand, and the tumour in the other, and attempting to glide them over each other. Anatomical knowledge helps the detection of infiltration inside a gland.

To the  
Vessels

Adhesions to **vessels** are detected by the immovable nature of the tumour in the longitudinal axis of the vessel affected, although the tumour may be movable over the skin, muscles, and bone, or may be moved transversely.

To the  
Nerves.

Adhesions to **nerves** and **neuromata** are detected in the same way as the above, that is its fixity in longitudinal axis of the nerve but its mobility to the transverse axis. Pain of focal or local nature helps us to corroborate the point.

(iii) Fluc-  
tuation.

(iii) **Fluctuations**.—The term means to a lay person anything which rises and falls in a way so that one reacts to induce the other. Commercial people watch the value of gold or currency or market commodities when it rises and falls, that is as it fluctuates. By

the term Fluctuation surgeons signify a special condition by which they understand that a swelling or a surface yields to pressure made by our fingers, but it at once rebounds and assumes its original level by **hydrostatic tension** induced by some fluid substance inside it, no sooner such pressure is released.

Fluctuation is a (a) Hydrostatic Tension.

(a) **Hydrostatic Tension**,—is a very important clinical sign, and it may require a good part of a life long experience to be cent. per cent. sure of the contents of the fluid. We are often required to stake our clinical experience basing on this sign alone.

The presence of Fluctuation indicates **presence of fluid**. But the phenomenon is not so easy to elicit by one hand as described above; and pressure exerted by finger on swellings containing gas or fat will also exhibit the same feature, that is of being soon followed by the resumption of the original level of the swelling. It is **impossible**

Fluctuation indicates presence of fluid.

to gauge the **hydrostatic** nature of the contents by simply pressing a tumour with the fingers of **one** hand only.

But it is impossible to elicit it by one hand only.

One should flatly place the balls of the fingers of one hand preferably of the left, with a little counter pressure, at one pole or side of the swelling, and then with the fingers of the other hand preferably of the right, gently press into the swelling against his left hand and then release the pressure, to be repeated alternately. If

The proper method.

during these latter acts a **rise** and **fall** is alternately felt under the fingers of the left hand corresponding to and synchronous with the pressure and release made by the right hand, this **sensation** is **fluctuation**. During this examination some precautions should be observed to avoid arriving at a fallacious conclusion, viz., (1) care must be taken to fix the tumour by the left hand by keeping a gentle compression or counter pressure on it, which should remain immovably on it, so that while the right hand which is kept free, is used to press on its other pole, the whole tumour may not move sideways "en masse." Such movement "en masse" is **mobility** of a tumour and not fluctuation, described above. This latter

Fluctuation is a sensation.

condition happens when both hands are moved. Fluctuation should be obtained in at least two inter-crossing axes of different poles, that is it should be observed by using the hands from side to side and then from off and near of a tumour; and if the contents are actually of fluid nature, the hydrostatic sign can be elicited from all directions. At any area of the tumour where solid degeneration or solid formation exists fluctuation would be absent. In elongated and narrow abscesses, or serous swellings, *e.g.*, abscess in a long compartment of a muscle bounded by septa, or in tenosynovitis, it is difficult to elicit fluctuation crosswise.

(b) Elasticity and (c) Compressibility.

(b) **Elasticity**, and (c) **compressibility** are quite distinct physical characters from hydrostatic tension; *e.g.*, fluids are least compressible and a fibroma may feel as elastic as an India-rubber toy ball. **Compressibility** is the property of being reduced in bulk by pressure, the contents of which may be brought or crowded together within narrower limits. Such a feel is of no important clinical significance. **Elasticity** on the other hand is the power of rebounding or restoring to original shape after compression; and this may be easily mistaken for fluctuation, although practically all fluctuating tumours are more or less elastic and compressible.

But the pitfalls are many.

Wrong detection.

Failure of right detection.

Not only the erroneous conclusions which a beginner may arrive at by faulty methods, but the pitfalls are many. One may conclude a swelling to possess fluctuation although there may be none, for the following reasons: (1) The swelling may be very loosely moveable. (2) The swelling may be very soft and compressible as a mass of fat. (3) The swelling may be elastic.

On the other hand one may fail to elicit fluctuation although the tumour may be actually a fluctuating one for the following reasons: (1) the hydrostatic tension may be extremely weak or entirely lacking; (2) the hydrostatic tension may be so strong that the swelling may appear to be quite hard and solid; (3) the depth of the accumulation may be so great, that it may be

difficult to examine properly owing to interposition and intervention of a large mass of soft tissues between the surface and the sac; (4) sometimes the small size of the swelling may render proper examination difficult.

The best corroborative examination to confirm fluctuation is to be sure of the **fluctuation-wave**. Fluctuation and Fluctuation-Wave are not signs of the same nature. A wave is determined by a **thrill** which the fluid may transmit from one pole to another if percussed. It will be described below under examination by Percussion.

Fluctuation in a swelling indicates that the swelling contains fluid, but the sensation gives us no hint with regard to the **character** of the fluid. It is a very important matter to rightly diagnose the nature of a fluid swelling to conduct its proper treatment.

The fluids in a swelling may be, (1) **exudates** as described in Volume I, Part II, Chapter III; (2), retained **secretions**; (3) obstructed excretions; or (4) necrotic degeneration, caseations, and softening.

To distinguish the various contents we should proceed as follows:—

(a) **Determine the anatomy** of the swelling, that is to say its actual seat. If it is in connection; (i) with any serous cavity, as a hæmatocele or hydrocele, the contents must be blood or some serous fluid, (ii) with any synovial cavity in connection with a synovitis or bursitis the contents are serous or mucoid, (iii) with any viscus as the urinary or gall bladder, the contents are urine or bile, (iv) with any secreting gland, *e.g.*, in hydronephrosis, mammary cyst, ovarian cyst, sebaceous cyst, the contents are serous, or consist of normal secretion of the glands concerned, (v) with any lymphatic gland, in acute condition the contents are pus, but with chronic fluctuating swelling the contents are of the nature of tuberculous or cancerous degenerations, (vi) with muscular septa the contents are pus as psoas abscess, (vii) with a joint it is a synovial cyst or tuberculous pus, (viii) with foetal tubular structure it is a dermoid or

Fluctuation wave.

How to determine the character of the fluid contents.

(a) Determine the anatomy

thyroglossal or other embryonic cysts, and the contents are serous or the physiological secretion of the tube concerned.

(b) Determine the history.

(b) **Examine the history.** *Vide* below. The nature and character of the fluid may to a certain extent be determined by the history of the case. The fluid varies according to whether the tumour is congenital, or one rapidly growing after an injury or inflammation, etc.

(c) Use the method of Percussion.

(c) Use the method of **Percussion**.

III. Percussion.

### III. PERCUSSION.

Next to the consideration of anatomical site and history, nothing else helps us to determine the nature of the contents better than **percussion**. We attempt to elicit the following clinical significance by the method of percussion, *viz.*:—

(i) Fluctuation Wave.

(i) **Fluctuation-Wave.**—This sensation elicited by percussion should not be confused with Fluctuation described under Palpation. Fluctuation-Wave is obtained in cystic accumulations of large size contained within more or less **tense** walls; *e.g.*, in ascites, or in unilocular ovarian cyst. This clinical sign is obtained as follows. The palm of the left hand is placed over one lateral aspect of the swelling without any evident pressure although kept in close contact with the surface. The fingers of the right hand are then used to tap sharply on the opposite pole of the lateral aspect of the tumour and played exactly in the same way as the Indians use the index finger on a *tabla*; that is to say the right palm is placed so, that the balls of the terminal portions of the third, fourth, fifth fingers, the thenar eminences, and the radial aspect of the thumb are placed in touch with the surface of the tumour, while the rest of the palm is flexed up a little above the surface. In these positions of the two hands, remaining in contact with the swelling at the same time, if

the index finger of the right hand is used to sharply tap on the surface of the tumour a corresponding wave could be felt distinctly, as it were to strike the palm of the left hand. It is a **wave** transmitted **through** the fluid medium of the tumour, and should not be mistaken with the mere impulse which may be carried along its surface. A fluctuation wave is a true gentle thud transmitted abruptly on the palm, and once felt can never be mistaken for an impulse. The best way to avoid the transmission of a surface impulse is to ask an assistant to place the radial edge of the palm of his hand on the summit of the tumour pressing on the surface as a sharp line of obstruction.

(ii) **Fremitus**.—A fluctuation wave differs in character with the consistence of the fluid inside the swelling. If a **fremitus** is perceived, as often occurs in Hydatids of the liver, such a sensation is accounted for the vibrations set up by the impact of the daughter cysts in the mother sac. This is described as **hydatid fremitus**. Auscultation over a hydatid during percussion sometimes reveals a musical sound.

(iii) **A Thrill** in a swelling of a point indicates the presence of solid bodies, as “melonseed bodies.” *Vide* Volume IV. Disease of the Joints.

(iv) **A Tympanitic** percussion note indicates the presence of **gas** inside a swelling. Small bubbles, or gas accumulated in some chambers, cannot be detected by this method, and the note is not elicited if the gas is not collected in sufficient proportion. Small accumulations are detected by their emphysematous nature.

#### IV. AUSCULTATION.

IV. Auscultation.

**Auscultation** is a very useful method of determining the nature of many **pulsating** tumours. Recently it has been very successfully employed in acute abdominal cases. By Auscultation we elicit the following main points :—

**A Bruit** heard over a swelling is due to, (a) Mural cause, that is rush of blood into an aneurysmal sac; (b) Extra-mural cause, such as partial compression of an artery by a tumour outside the vessel. Such a rush is heard synchronously with the heart, and simultaneously with the pulse.

A Bruit is described according to the character of the sound heard by such terms as soft, loud, blowing, musical, rough, dull, or like a thud, etc.

The points which are required to be noted while hearing a Bruit are, (a) the time when it occurs, (b) can it be heard in uniform intensity at every area throughout the whole swelling? (c) Can it be obliterated by compression on the proximal part of the artery? (d) Is it intensified by pressure? (e) Can it be heard at a distance, being only conducted by a diseased artery?

Aneurysmal bruit is of a blowing character, but bruit in partially compressed vessels yields a dull, toneless thud. An aneurysmal bruit is not intensified by pressure, but a bruit in a partially compressed vessel is intensified if the pressure is applied in the direction of the artery. An aneurysmal bruit is equally heard all over the swelling but an arterial bruit is heard loudest on the line of the vessel only. An aneurysmal bruit is conducted along the affected artery, and is heard at a distance but a compression bruit is not conducted at a distance.

Auscultation is a very useful method of diagnosing or differentiating a gravid uterus from uterine and ovarian tumours. Foetal heart gives a clear 'tic' 'tic' sound as heard from a watch underneath a thin pillow.

In acute abdominal cases, at the initial stage, we can very nearly make a correct diagnosis between peritonitis and acute Obstruction by means of Auscultation. In obstruction a distinct rumbling sound of loud and irregular peristalsis is heard under stethoscope, if placed especially on the proximal part of the obstructed gut. If under a stethoscope we hear sounds, resembling those

we hear on drawing a wet finger across the surface of a wet rubber sheet which gives sounds of a rhythmic nature, coming out roughly at the rate of two times a second, the condition demonstrates a weak and sluggish peristalsis underneath. This rhythmic sound is present in the other quadrants of the abdomen in contrast to its absence and the consequent complete silence at the areas where peritonitis has supervened.

At this stage of our investigation we are especially face to face with the question whether the tumour contains solid, fluid, or gaseous, substance or a combination of two or all of them. We may be expected to learn at this stage to determine the following points:—

Now determine the nature of tumour.  
Fluid  
Gaseous  
or Solid.

(i) The presence of fluid inside a swelling. This we know is diagnosed by:—

- (a) Pitting on pressure.
- (b) Fluctuation.
- (c) Fluctuation-wave.

(ii) The presence of gas inside a swelling is determined by:—

- (a) Fine crackling.
- (b) Crepitations.
- (c) Emphysematous condition.
- (d) Tympanitic percussion.

All the above vary according to the nature of the gas formed.

(iii) Solid tumours are recognized by:—

- (a) Elasticity.
- (b) Dull percussion.

The solid tumours may be sub-divided into: (1) Those which are compressible and soft, *e.g.*, lipoma, a condition which should be carefully distinguished from fluid or fluctuation in a swelling. (2) Those which are compressible and elastic but not of bony hardness, *e.g.*, adenoma, fibroma, sarcoma. (3) Those which are absolutely hard, *e.g.*, osteoma, enchondroma, etc.

## V. Mensuration.

## V. MENSURATION.

When the **nature** of the tumour is determined and its contents to a certain respects ascertained, as well as its **form** carefully observed, by means of the four previous methods of examination described above, *viz.*, Inspection, Palpation, Percussion, and Auscultation we should never forget, especially in cases of general abdominal swellings, to **measure** their physical extension and size. The measurement of the circumference of a swelling may be taken over the summit of the swelling at places of the body other than abdomen; but on the abdomen it should be taken at the level of the umbilicus, or at the equator of the maximum distension. The measurement should be taken at least in two different positions of the patient, *e.g.*, while the patient is lying and then again while sitting.

## VI. Clinical Characters of Tumour.

## VI. CLINICAL CHARACTERS OF TUMOUR.

If any person is appearing for any examination or scrutiny or trial, a court as a rule demands of him to submit his birth certificate and lay all other informations before it regarding his **integrity**; show them his past conduct report, or the progress report of the work done by him and the way he has made such progress as well as the effect of his achievements on himself, on his neighbours, and on his community at large. The same procedure may be followed in our clinical investigations. Having finished the physical characters of a tumour and having formed some qualified opinion about its diagnosis let us now **hear** its: (i) **life history**, (ii) **progress**, and (iii) its **sequelæ**.

## History and the vital characters of a tumour.

## Clinical history.

(i) In considering the **clinical history** of a tumour the following factors demand our notice. *Viz* :—

(1) How did it make its first appearance? In other words, let us enquire and investigate its mode of **origin**.

A tumour may be: (A) Congenital or (B) Acquired. The latter may be sub-divided into **four** groups, namely, (a) **traumatic**; (b) **inflammatory**; (c) **neoplastic**; (d) and those produced by some **obstruction**. If the origin of the tumour is known we can then determine the group to which it belongs. We can next enquire of the **progress** of its development, its **sequelæ** and effects its progress has made: (i) on the neighbouring tissues; (ii) on the whole organism.

A. Congenital.  
B. Acquired.  
Acquired tumours may be:—  
(a) Traumatic.  
(b) Inflammatory.  
(c) Neoplastic.  
(d) Obstruction.

No sooner we are able to determine to a certain extent whether a tumour is of **fluid**, **gaseous**, or **solid** consistence by means of the methods previously described its further investigation so far as its **origin** is concerned demands our notice from two view points only: *viz.*, (i) Did the swelling arise **suddenly** and was it formed **rapidly**; or in other words is it **acute**? (ii) Or did it form **slowly**, or in other words is it **chronic**? Some of the latter forms of tumour may be **intermittent**. Let us examine its primary vital character regarding its origin in greater detail first. We shall then take up the consideration of its **progress**, and **sequelæ**, as far as these factors would help us in our diagnosis at a later stage of our investigation.

No sooner we know the consistence of the tumour whether fluid or Gaseous or Solid; and no sooner we are aware of its origin we need to consider it from two view-points:  
*Viz.*,  
(i) Is it Acute or  
(ii) Chronic?  
Some may be Intermittent.

The next point of vital importance the surgeon is to consider, is to determine if a tumour is a **pulsating** swelling or a **non-pulsating** one. The correct diagnosis of a tumour manifesting any impulse corresponding to the heart beats is a life and death question. Such a tumour may be exhibiting the pulsations **primarily**, that is to say the tumour having a direct communication with the blood current; or, **secondarily**, that is only the impulse of the pulsation of the circulation is being

Is it pulsating or Non-pulsating.

transmitted, owing to the situation of the tumour being in contact with or at the adjoining neighbourhood of a main blood vessel.

Pulsating tumours may be soft or hard, and they may be in association with any groups of swellings, *viz.*, (a) Traumatic; (b) Inflammatory; (c) Neoplastic; and (d) those due to obstruction. We shall presently return to the point.

It would be a little too tiring for a beginner to go through the description of a long list of surgical lesions with reference to their diagnosis. We shall therefore set them out in a Table at first to help his memory more rationally; and in a way which may be useful in proceeding with a case systematically. *Vide* Table XXII.

We shall enumerate the tumours with reference to the above Table in a greater detail which follows:—

#### A. Congenital tumours.

**A. Congenital tumours.**—The congenital tumours may also be divided into cystic and soft tumours, and solid or hard tumours. They may be sub-divided under three groups. *Viz.*: (a) Traumatic—*e.g.*, intra-uterine injury, *viz.*, dislocation or intrauterine fracture with thickenings around bones. (b) Neoplastic—*e.g.*, fibroma, lipoma, sacral tumours, etc. (c) Malformations,—*e.g.*, spinabifida, meningocele, encephalocele.

#### B. Acquired Acute, Fluctuating and Non-pulsating.

**B. Acquired tumours. Soft.**—A soft, cystic, fluctuating, tumour, **non-pulsating** but arising suddenly:—

(a) If traumatic, is **effusion**, which may consist of: (i) blood that is hæmatoma, when forming rapidly, or, (ii) lymph when forming slowly.

(b) If inflammatory, is **exudation**, and the accumulated fluid may consist of, serous, hæmorrhagic, or purulent matter, ultimately forming into an **abscess**.

(c) If neoplastic, is usually a degeneration inside an encephaloid carcinoma or sarcoma, or growths in direct communication with extravasation.

(d) If the swelling is due to obstruction, such a condition may arise from three different factors, *viz.*, (i) obstruction to a **duct**, such as cysts arising from





retention or distension; *e.g.*, salivary obstruction giving rise to a tumour in the mouth or the neck, after meals in salivary calculus; (ii) obstruction to **lymphatics**; (iii) obstruction to **vessels**; which are usually venous, or other aneurysmal accumulations.

Retention of urine due to stricture resulting in an acute tumour at the hypogastrium, is also an instance of acute obstruction to the flow in the passage. Milk congestion in the lacteals is another similar instance. Such a distended viscus, resembling a tumour is recognized by, (a) its anatomical position, (b) the signs of obstruction to its secretion, or excretion, *e.g.*, of saliva during or after a meal, or retention of urine, (c) the effect of removing the contents, *e.g.*, disappearance and collapse of a distended bladder after catheterization of the bladder in retention of urine, (d) the outline of the swelling corresponding exactly to that of the viscus concerned.

Acquired **pulsating** swellings of fluctuating nature arising suddenly may be:—

(a) If traumatic, a rapid effusion of blood, which may be due to ruptured artery or ruptured aneurysm. A ruptured artery is associated with no history but is a direct effect of trauma, whereas a ruptured aneurysm has a previous history of the formation of an aneurysm, and may arise spontaneously without any trauma or from a slight violence only.

(b) If inflammatory, it is due to an effusion establishing direct communication with many capillaries, or a main artery, or vein. Suppuration in the neck is often associated with such grave complications, and it is sometimes difficult without very careful examinations if the pulsation in an abscess in the neck is due to mere transmission of a thrill of an underlying uncomplicated vessels, or to direct pulsation of an open vessel inside it.

(c) If neoplastic, the pulsating swellings are usually sarcomatous, and a sarcoma sometimes starts to pulsate suddenly.

Acquired,  
Acute,  
Fluctuat-  
ing, and  
Pulsating.

(d) If an acute fluctuating swelling is due to any obstruction to vessels, or to aneurysms, or cyst formation, without any evidence of trauma or inflammation, and if it starts to **pulsate** such a pulsation may be produced by sudden extravasation, or congestion.

Chronic  
Fluctua-  
ting and  
Non-  
pulsating.

A tumour developing slowly and having the consistence of fluid nature but **non-pulsating**:—

(a) If traumatic, is due to **effusion** or hæmatoma slowly formed. Synovitis may develop in the same way. Effusion of plastic lymph may give rise to the same kind of swelling. Chronic Arthritis may be excited to effusion by injury.

(b) If the swelling is inflammatory, is due to necrotic degenerations, *e.g.*, tuberculous abscess, gumma, etc.

(c) If it is a chronic fluctuating non-pulsating neoplasm, is due to cystic degenerations.

(d) May be an obstruction cyst, or a blood cyst being formed from various causes, *e.g.*, all retention or distension cyst.

Cystic swellings due to retention and accumulation of secretion is diagnosed; (i) by its practically globular outline; (ii) by the fluctuation and fluid nature of the contents; (iii) by the absence of any evidence or signs of inflammation; (iv) by its position corresponding to a gland or its being situated actually on a gland or inside it or a closed sac; (v) by the determination of the fluid nature of its contents by exploratory puncture; (vi) by the absence of pulsation or absence of any connection with vessels.

Chronic  
Fluctuating,  
Pulsating  
swellings.

Acquired **pulsating** tumours developing slowly are usually Aneurysms or Pulsating Sarcoma. Such development may be due to traumatic, inflammatory, neoplastic or other aneurysmal causes. *Vide* Aneurysms.

There are some indications by which aneurysmal swellings could be more definitely recognized which are the following:—

**Dilation of an Artery** or formation of an Aneurysm

is evidenced and diagnosed: (i) by its position on the line of the artery; (ii) by its fixed nature of position in connection with or associated with the artery; (iii) by its pulsating and expansile nature, the pulsation being synchronous with the cardiac systole and often accompanied by thrill or bruit; (iv) by the nature of the pulse in the distal aspect of the implicated artery which is weakened or delayed.

**Dilatation of a vein** on the other hand is diagnosed; (i) by the position, on the line of the vessel or sometimes at stagnant places produced by obstruction at the proximal aspect or action of gravity or posture. It is therefore most common at the lower limb, anus and spermatic cord; (ii) by the tortuous or elongated or sometimes sacculated character of the swelling; (iii) by its reducibility and its compressibility both of which can be effected by directly compressing on the swelling or at the distal aspect of the swelling; (iv) by its pulsation and livid colour of the swelling which happens when direct communication is established between the vein and an artery especially when superficial.

Non-fluctuating swellings may also form primarily in two ways, *viz.*, (i) rapidly developing or **acute** forms; (ii) slowly developing or **chronic** forms.

A rapidly forming soft non-fluctuating swelling:—

(a) If traumatic, is an **infiltration**, or an **extravasation**, *e.g.*, Acute CEdema, Gas Infiltration, Emphysema of the neck, Extravasation of blood. Acute soft infiltrations associated with traumatic history are usually of cedematous origin and their development is due to obstruction of the lymphatics or veins. Extravasation of urine is an instance of urine infiltration, and is detected by the history of stricture, retention of urine which is suddenly abated without any evidence of micturition, and anatomical limit of extension at the perineum. Extravasation of blood is diagnosed by: (i) discolouration of the part which cannot be altered by pressure, (ii) the changes the discolouration exhibits afterwards, (iii) the

ill-defined nature of the swelling, (*iv*) by the history of trauma.

(*b*) If inflammatory, is due to spreading diffuse infiltration of the infection, *e.g.*, Cellulitis.

Infiltration due to infection may produce soft non-fluctuating swellings in tissues as well as in lymphatic glands, *e.g.*, tonsils, adenoids, etc. Infiltration may produce the same effect under the periosteum, larynx, tunica vaginalis, synovial membrane, bursæ, etc. An infiltrated gland is diagnosed by, (*a*) its position; (*b*) its outline corresponding exactly that of the gland or organ; (*c*) the signs of obstruction to the secretion if there is any.

(*c*) If neoplastic, soft swellings may be a sign of implication of glands due to the extension of malignancy.

(*d*) If aneurysmal in origin, it may be a diffuse aneurysm, or an aneurysm getting solidified. Thrombosis in vein may produce swelling at the distal part of the limb the nature of which is cedematous. Lymphatic obstruction forming scrotal tumour, acute elephantiasis, etc., are other instances of tumours resulting from lymphatic obstruction, and passive cedema. All types of cedema, *viz.*, cedema due to lymphatic obstruction, cedema produced by capillary dilatation due to inflammation, cedema due to venous obstruction, and cedema due to arterial or cardiac condition, pit on pressure in the same way. Inflammatory cedema is associated with pain, redness, heat, and tenderness, and is more or less confined to a definite area. Lymphatic cedema can be entirely obliterated at any spot by firm and continued pressure. When cedema is due to venous obstruction, it is limited into an area, and there is generally more or less lividity of the surface and distension of the veins. Cedema due to altered condition of the blood is usually distributed at different areas of the body, and there are signs of pallor and anæmia. Extravasation of urine could hardly be confused with cedema.

Non-fluctuating soft swellings may be suddenly produced by strangulated hernia. History, mode of its formation, anatomical position, and acute obstructive signs of strangulation of the gut are the points for its diagnosis.

Pulsation in a non-fluctuating soft tumour is produced in sarcomatous condition, or diffuse aneurysm; in the latter condition the pulsation is faint. Any of the preceding conditions may be associated with the transmission of cardiac impulse by being situated on a main vessel lying immediately subjacent to it.

A non-fluctuating swelling slowly formed may be:—

(a) If traumatic, an infiltration organized into fibrosis.

Non-fluctuating swellings slowly formed.

(b) If inflammatory, it is a thickened œdema. Chronic synovitis, hypertrophy of the mamma, tonsils, etc., are also such instances. Hypertrophy can be easily recognized, by its especial features, *viz.*: (i) by the non-interference of the function of the organ or tissue involved, excepting some mechanical hindrance produced due to bulk; (ii) by the painless nature of the swelling; (iii) by the ceasing of the progressive nature of the growth after attaining a certain dimension; (iv) by the absence of signs of inflammation; (v) by the absence of any degeneration or retrogressive changes.

(c) If neoplastic,—it is a soft lipoma, or myxoma, or encephaloid cancer.

(d) If due to obstruction to the circulation, or duct, it may be produced by the alteration in the character of the blood, or obstruction to the lymphatic and venous circulation causing chronic œdema, *e.g.*, scrotal tumour, elephantiasis, etc.

Bubonocoele, and reducible hernia appear like soft swellings at the groin. Site, history, and impulse, help us to diagnose it correctly.

Young women sometimes exhibit local signs of hysterical uterine swellings, produced by imaginary fright or idea tumour.

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of the existence of a physiological or a pathological growth; and its resemblance with soft non-fluctuating swellings on the uterus, at the hypogastric region, such as myoma and pregnancy, becomes so pronounced that it becomes exceedingly difficult by physical examinations alone to come to a diagnosis. The tumour miraculously disappears on putting the patient under chloroform. Imaginary pregnancy of young married sterile women who are anxious to have a child is also of the same nature. Diagnosis rests on the determining signs of pregnancy under chloroform as many signs such as amenorrhoea, etc., are actually present.

Hard swellings.  
(i) Elastic and hard.  
(ii) Bony hard.

**Hard swellings** may be considered under two groups, *viz.*, (i) Those which resemble the consistence of India-rubber that is **elastic** and **hard**; (ii) Those which have the density of **bony hardness**.

Both these types may arise suddenly or rapidly, and slowly.

Hard acute swellings of elastic consistency.

A hard swelling arising rapidly but of **elastic** consistency may be:—

(a) If traumatic, a *displacement* which can be easily recognized by the following signs: *viz.*, (i) the disappearance of the part or organ from its normal anatomical site; (ii) by the character of the swelling resembling the outline of the lost part or the dislocated organ, (iii) by the continuity of the swelling with the organ or part supposed to be dislocated.

(b) If inflammatory, it is an *exudation*, *e.g.*, under the periosteum, or an arthritis, etc.

(c) Neoplastic swellings of elastic consistence cannot form so acutely. They are usually sub-acute.

(d) If due to obstruction, it is usually a rapid distension of a cystic tumour.

Hard chronic swellings of elastic consistency.

A hard swelling arising slowly but of **elastic** consistency may be:—

(a) If traumatic, it is a hypertrophy.

(b) If inflammatory, it is a hypertrophy, chronic arthritis, etc.

(c) If neoplastic, it is a scirrhus cancer, or osteochondroma, or sarcoma.

(d) If due to obstruction, it is a hard cystic distension.

A hard swelling growing acutely and of **bony** consistency may be:—

(a) If traumatic, it is a dislocation when on the joint, and a fracture when on the long axis of the bone.

(b) If inflammatory, it is osteomyelitis, or arthritis.

(c) Neoplastic tumours growing rapidly cannot be of hard consistence.

A hard swelling growing slowly and of **bony** consistency may be:—

(a) If traumatic, it is a hyperostosis, callus, or recurring dislocation.

(b) If inflammatory, it is a spinal curvature, Pott's disease, neuropathic arthritis with dislocation, etc.

(c) If neoplastic, it is an osteoma or an enchondroma.

We may recapitulate the above points set out in the Table No. XXII.

### (3) Progress of a Tumour.

(3) The progress.

If it is possible to watch the course of the progress of a tumour it gives us useful indications as to its pathological nature. A tumour may change in various ways, namely, (i) **increase** in size, (ii) **diminish** in size, (iii) remain **stationary**, (iv) change its **position**, and (v) may change the **direction** in which it enlarges. We shall describe these features in greater detail below:—

(i) Increase may take place in size, but the **rate** of increase varies a good deal in different ways as follows: (a) **Suddenly** increasing in size, and then remaining stationary or the progress remaining arrested by surrounding resistance: such a change is usually due to

(i) Increase in size.

(a) Suddenly.

- acute inflammatory proliferation, or exudation in or around it. The proliferation may be associated with cellular exudation or fluid effusion, or extravasation. A hydrocele may turn into a hydro-haematocele and increase in size. A hernia may get strangulated, and increase in size. (b) **Progressively** increasing in size without any sign of arrest of such a progress, but the rate of increase may vary; *e.g.*, a continuous enlargement of a solid tumour indicates the nature of a malignant neoplasm. When it increases rapidly it may be malignant infiltration, or acute cyst formation, or inflammatory exudation. When its rate of increase is feeble it is usually due to resistance of the surrounding tissues, *e.g.*, subperiosteal or interosseous inflammations, and tumours, aneurysms, etc. (c) **Intermittently** increasing, *e.g.*, displacement of a viscera, and its replacement as in hernia. Successive attacks of acute and repeated inflammation. Intermittent filling and emptying of some vascular tumours, *e.g.*, exophthalmic goitre, varicose veins during pregnancy.
- (b) Progressively.
- (c) Intermittently.
- (ii) Diminish in size. (ii) May **diminish in size**.—Sudden reduction indicates evacuation inside in the body cavity or tissues, or rupture followed by the escape of contents into a body cavity, *e.g.*, rupture of an ovarian cyst, or acute obstruction of gall bladder with swelling followed by its complete absence produced by rupture of the viscus, or the clearance of the bile *via* the duct. Gradual reduction indicates absorption, and is a favourable sign in acute or chronic inflammation.
- (iii) Stationary. (iii) May remain **stationary**.—This usually happens in non-malignant neoplasms.
- (iv) Change its position. (iv) May change its **position**.—Lipomata may travel a long distance. Psoas abscess may show its appearance at various aspects of the body. Hernia exhibits many changes of position, *e.g.*, from groin to scrotum, or into the abdominal cavity.
- (v) Change its direction. (v) The **direction** in which a tumour extends and increases in size, is sometimes an indication which is of much value to arrive at a diagnosis. Such a change

occurs when the swelling is influenced by gravitation or encountered by least resistance. Psoas abscess, iliac abscess, extravasation of urine, diffuse lipoma can be easily and correctly diagnosed by the track or their passage they adopt to extend and enlarge. Lymphatic extension takes the course of lymphatic circulation. Varix, aneurysm, thrombus, may all be similarly spotted by their directions and anatomical positions. Synovitis, teno-synovitis, cellulitis, bursitis are other instances.

The examination of the **effects on the neighbouring tissues** is an important part of the methods of diagnosis.

Effects on neighbouring tissues, and the ways of extension. The three ways of extension.

The ways of extension are chiefly three; *viz.*, (a) by continuous displacement by the expansion of the swelling, (b) by infiltration of the tissues around it, (c) by colonization at a distance. Non-malignant tumours expand as stated first. Inflammation and malignant tumours infiltrate, and general sepsis and malignant metastases colonize.

#### **Effects on the particular tissues :—**

(1) Effects on the **nerves**, and their diagnostic value :—

(1) Effects on the nerves.

Pain caused by any swelling is due to four factors, *viz.*, (a) **Infiltrating nature of the toxin** of the exudation irritating the nerves; and in the case of tumour, implicating the nerve trunks, or the nerve terminals. When the latter are involved it is local. When the trunk is involved it is referred or focal. In acute inflammation a local pain is always present and the pain increases with any condition that excites vascular engorgement; as such engorgement increases the tension inside the body of the swelling, or excites traction on the tumour. Passive obstruction to the blood or lymph flow, exudation in closed bony cavities, twist of a pedicle of an ovarian tumour, are the instances of the different conditions described above. Slow persistent expansion produced by displacement of tissues is as a rule painless. (b) The **seat** of a swelling developing on a sensitive part or a nerve trunk may be a primary factor to produce pain. (c) The

**hydrostatic tension** of the fluid either caused by rapid and profuse effusion, or by the non-resisting character of the surrounding wall or tissues is a constant factor to produce pain. (d) The **traction** on the tumour produced by mechanical causes as gravity or torsion gives rise to pain. All these causal factors and effects give valuable indications as to the nature of the swelling.

(2) On the distal parts. Pressure symptoms, and muscular spasms.

(2) Effects on **the distal parts** :—

(i) **Pressure** symptoms may be evidenced by (a) oedema of the part distal to the swelling, (b) nutritional disturbance of the same part, (c) with concomitant paralysis, and (d) atrophy.

(ii) **Muscular spasm**.—Sometimes in acute condition a swelling may become the immediate cause of death, *e.g.*, on the larynx. Bell's paralysis from malignant growth on the parotid, or paralysis of half the tongue from aneurysm of the carotid, are other instances of spasms of the muscles. Muscular spasm is a prominent feature of Pott's disease. Usually paralysis sets in after irritative spasms, which in time is followed by atrophy.

(3) Effects on the muscular and con. tissues.

(3) Effects on the **muscular** and **connective** tissues :—

(i) Atrophy

(i) **Atrophy** of the tissues produced by stretching, thinning, absorption, expansion is a valuable guide to diagnose many tumours.

(ii) Deformity.

(ii) **Deformity** of bones, joints, spinal column, or expansion of bones, *e.g.*, of facial bones, are other signs which may aid to our diagnosis.

(iii) Fracture.

(iii) **Fractures**, after a little violence, or occurring spontaneously, establish the diagnosis of many sarcomatous and myeloid tumours, or pressure atrophy caused by aneurysm or other aggressive growths.

(4) Effects on the vascular system. (i) The arterial pulse.

(4) Effects on the **vascular** system :—

(i) The **arterial** pulse on the distal side may be greatly affected by the presence of some tumours, *e.g.*, if the pulse beats are of less tension and are smaller or

delayed than in the similar artery at the other limb the tumour is possibly an aneurysm. A sphygmographic tracing should always be taken. If the pulse is entirely lost the vessel is blocked by embolism or sometimes thrombosis inside the vessel, or extramural pressure on it from outside, or the tumour obliterating the flow by direct invasion. So long as a pulsating tumour pulsates itself the distal artery will pulsate. Extravasation or hæmatoma with complete subcutaneous rupture of the artery or rupture of an aneurysm is evidenced by abolition of the arterial pulse of the vessel concerned beyond the lesion. A tumour near an artery may displace it, or compress it but if the artery is not involved it can hardly occlude its lumen. A very vascular pulsating growth, gives no evidence of alteration of the pulse. A transmitted pulse-thrill of the arterial circulation may be communicated through a tumour, but in that case the pulse is unaltered which a sphygmographic examination will clearly reveal.

(ii) **Venous** engorgement is evidenced by the dilatation and tortuous coursing of the veins over the tumour. By the general change of colour into purplish or livid hue of the surface a clear indication is given of the tumour being in close implication with the venous system. Venous distension may be produced by various causes. *Viz.* : (a) The swelling may compress or obliterate the vein or veins and thus produce distension by mere mechanical pressure. (b) By communication between an artery and a vein, the force of the arterial flow may be impeding the venous return. (c) The venous distension itself may be the primary cause of the swelling appearing like a dilated, sacculated, or convoluted tube. But this can be entirely reduced in a case by obliterating the back flow by pressure at the distal side of the swelling, unless the blood is thrombosed or solidified inside the vessel. (d) The distension of the vein and the formation of the swelling may be due to the same cause of engorgement, *e.g.*, heart disease and œdema, the distension being

(ii) The venous engorgement.

due to obstruction or regurgitation in the cardiac and pulmonary circulation. (e) The distension may be due to increased supply, necessitating enlargement of the efferent veins. Very vascular new growths of malignant nature, or vascular benign tumours are the instances; the distension being usually observed on the cardiac side of the swelling, and it usually happens where the original calibre of the vessel is not sufficient to carry back the large amount of blood propelled into the swelling.

(iii) Lymphatic  
œdema.

(iii) **Lymphatic** obstruction when caused suddenly as a result of obliteration of lymphatics, or veins, produces acute œdema which pits on pressure easily, and is marked by varying lividity. The most peculiar clinical exceptions are, at the root of the neck and groin. At these places œdema produced by malignant diseases, *e.g.*, at the root of the neck by growths in the axilla, and at the groin, seldom exhibits pronounced lividity, and the skin becomes soon thickened and irregular; and in some cases the œdema is very firm and does not yield to pressure, or at best pits with difficulty.

The effects  
on the  
general  
system  
vary ac-  
cording to  
different  
kinds of  
swellings.

The effects on the general system vary according to the different kinds of swellings, which are described under different pathological conditions. The general **cachexia** usually observed in patients is important in this sense only, that it indicates an extension of the poison throughout the system and is therefore of great omen. Otherwise so far as arriving at a particular diagnosis of the cause or the nature of the swelling is concerned, **cachexia** is of little importance. It only gives a corroborative evidence of the malignant nature of the swelling. Cachexia is the result of malignancy and not the cause of it, or of the tumour, although cachexia produced by some less harmful diseases may induce or predispose a more fatal malady.

VII. Corro-  
borative ex-  
aminations.

## VII. CORROBORATIVE EXAMINATIONS.

In addition to the above Physical Examinations for diagnosing the nature of swelling by which in most of

the instances we may be able to arrive at a correct diagnosis, many other corroborative methods help us in arriving at a correct conclusion in most of the remaining doubtful cases. The methods employed are the following :—

1. General Clinical Observation: *e.g.*, (i) age, (ii) sex, (iii) occupation, (iv) concomitant affections, and terminations.

2. Surgical exploration and other minor operations.

3. X-rays examination.

4. Microscopical examination of pathological tissues and discharges.

5. Bacteriological examination.

6. Examination of the special senses by special instruments. *E.g.*, **Tuning Fork** for ear trouble, in errors of refraction by the uses of eye instruments.

#### 1. General Clinical Observations, *viz.* :—

(i) **AGE**.—Experience teaches us that consideration of age is a very important diagnostic help to determine the nature of the swelling. We pass one mile stone after another till we finish the journey of our life. In this path of life the pitfalls are many, and the accidents and troubles met with in the way of our travel are of the natures which are quite different according to the distance of the road travelled and therefore characteristic of the age.

1. General clinical observations.  
(i) Age.

(a) Swellings of **infancy**.—We come across with the following kinds of tumours and swellings immediately after birth, *viz.*, *growths* such as *angioma*, *nævi*; *cysts*, such as *dermoids*, *cystic hygroma*; *malformations* such as *spina-bifida*, *meningocele*, *encephalocele*, *hydrocele*, *hernia*, etc. It is in the first year of life rickets, scurvy-rickets and syphilis are met with. In the second year rheumatic nodes, occasionally fatty tumours, and sarcomata are found, neoplasm seldom or never occurs in the first year of life.

(a) Swellings of infancy.

(b) Swellings of youth.

(b) In the childhood, or **youth** excess or proliferation of lymphoid tissue gives rise to glandular enlargements, *e.g.*, hypertrophied tonsils, abscess, acute and chronic, osteomyelitis, cartilaginous and bony tumours, and all kinds of tuberculous and traumatic swellings.

(c) Swellings in early adult life.

(c) Swellings in **early adult life**.—All kinds of traumatic and acute inflammatory swellings, chronic syphilitic or venereal granulomata, and various kinds of fatty and mucous tumours, hydrocele, orchitis, and sarcomatous growths, make their appearance at this period of life. Obstruction cysts, aneurysm, and varix also appear at the latter part of this period of life.

(d) Swellings in **late adult life**,—are mostly of malignant and senile nature, *e.g.*, carcinoma, rheumatoid arthritis, etc.

(ii) Sex.

(ii) **SEX**.—Various neoplastic growths show a sex predilection, *vide* ætiology of Neoplasms, Chapter II. Otherwise sex has little or nothing to help in diagnosis.

(iii) Occupation.

(iii) **OCCUPATION**.—Very often its consideration gives us direct and useful hints in diagnosing many swellings, acute or chronic, professional or traumatic.

(iv) Concomitant affections, previous history of the patient and later history of the swelling

(iv) **CONCOMITANT AFFECTIONS**, previous history of the patient, and **later history** or **terminations** of the swelling, should be carefully studied to arrive at an exact diagnosis. Concomitant factors associated with a swelling simplify diagnosis to a great extent; *e.g.*, presence of pediculi in the head and beard will determine the diagnosis of a few neck glands not being due to tuberculosis, the doubt of which might become a fruitful source of anxiety. A few syphilitic patches somewhere else in the body will fully establish the diagnosis of a gumma otherwise doubtful. The association of exophthalmos with goitre is another instance. A gland at the groin may direct us to proceed on a different line of a treatment if we can detect a small injury at the toe; or something wrong at the anus.

As the **previous history** of the origin of the swelling

greatly determines its diagnosis, so also the previous history of the patient himself and his parents helps us to recognize the nature of many swellings. There are factors which by heredity or subsequent habits and circumstances establish a predisposing condition of **diathesis**, or gives us a definite clue of determining our diagnosis; *e.g.*, hæmophilic swellings, syphilitic diathesis, or circumstances such as recurrence of a growth after a previous extirpation in carcinoma, etc.

**Later History** of the swelling or its **terminations**,—if it can be watched without harm to the patient, helps us greatly towards right diagnosis and therefore leads us to right treatment. This might be observed particularly by the following features:—

(i) Evidence of **infectivity**.—Swellings exhibiting distinct evidence of local or general infective processes, especially when they extend, their particular mode of infiltration or extension, *via* the direct continuity of cellular tissues, or lymphatics, or circulations, are very important diagnostic features. (i) Evidence of infectivity.

(ii) Evidence of **malignancy**.—Swellings exhibiting distinct evidence of local malignant infiltration or general dissemination of malignancy, and the particular mode of local invasion or infiltration or general dissemination *via* the lymphatic as happens in carcinoma, or *via* the veins as happens in sarcoma, are very important diagnostic features. (ii) Evidence of malignancy.

(iii) Evidence of **local pathological changes**.—The changes described above which are manifested by infective or malignant features may be observed by various local pathological terminations; consisting mainly of **alteration in consistence**, which may subsequently become harder or softer, and **ulceration**. Unless we are sure of the nature of the change, either of the two, *viz.*, harder or softer, may be the cause of either the surgeon's grief or pleasure, or neither. For instance, if an indurated chronic swelling would exhibit signs of softening it may be due to **resolution** or **suppuration** or **degeneration**; and

such an indefinite situation would only induce ignorance to prognosticate or sophisticate, or adopt an uncalled-for optimistic view. A pyococcal inflammatory induration, tuberculous induration, or a malignant induration, has each its own way of termination by softening; and what is a favourable sign in one case is a sign of anxiety in the other. (1) **Softening of a swelling** may take place in the following circumstances: (a) Liquefaction, acute necrosis, and suppuration. (b) Chronic degenerations of the nature of necrobiosis. (c) Absorption of effusions. (d) Atrophy or absorption of hardened exudation, or fibrosis. Similarly **hardening** or **increased firmness** may be due to: (a) Increased hydrostatic tension by increased exudation or effusion counteracted by the resistance of the firm neighbouring tissues. (b) Increased displacements or herniated condition. (c) Obstruction to venous or lymphatic back flow by twists, etc. (d) Organization produced by resolution, coagulation, or calcification; fibrosis of the fluid contents, and effusions; solidification of blood, exudation or extravasation in a hæmatoma, aneurysm, and varix; callus-formation, ossification, or osteoid sarcomatous changes, etc. In the cases of resolution and progressive regenerative organization and callus-formation they are followed by atrophy and absorption, but, osteoid formation is a progressively degenerative or neoplastic phase. (2) Softening produced by necrotic degenerations may extend towards the surface, disintegrate the covering parenchymatous tissues, and affect the surface ultimately ending in **ulceration**; and granulomatous or neoplastic proliferations. Friction or injury on the surface of a deep-seated tumour may induce the same terminations.

## 2. Diagnostic surgical operations

## 2. Diagnostic Surgical Operations.

In many instances the difficulty of arriving at a diagnosis becomes insurmountable. Before we undertake

any method of radical operation for these doubtful cases, minor operations are done for diagnostic purposes. *Viz.*—

(i) Exploration.—This is done more to tap the nature of the contents of a cystic cavity. This can be done by puncturing the swelling by a sterilized hypodermic needle and sucking up the contents in the syringe, or by an aspirator. *Vide* Minor Surgery.

(ii) Incision.—This is done by a simple incision down to the growth. It is employed to know the exact nature of the consistence of a soft non-fluctuating or an elastic swelling. It also gives us an idea of the actual seat of the swelling.

(iii) Slicing off a little tissue.—A little portion of the tissue may be cut out and submitted to microscopical examination.

All the above procedures are very dangerous, and should on no account be lightly undertaken by a junior in the science. Regarding their drawbacks, the reader is referred to Minor Surgery. They may extend the trouble, or light up to more rapid proliferation, or the lesion may get generalized from a local condition.

(iv) Catheterization—requires no further explanation.

(v) General anesthesia, *e.g.*, gas, ether and chloroform may be employed to establish the diagnosis of many neurotic swellings, imaginary uterine tumours, fancied pregnancy, and difficult or painful tumours.

### 3 & 4. Microscopical and Bacteriological Examinations.

The above diagnostic surgical operations are useful not only from the point of view of macroscopical or naked-eye examination, but the parts removed may be submitted to microscopical examination. The discharges may be examined by bacteriological examinations by staining, and culture methods, and **inoculation** into animals. *Vide* Diagnostic Clinical Methods. Chemical

3. Microscopical and  
4. Bacteriological  
Examinations.  
Inoculation.  
Chemical examination, &c.  
are helpful to arrive at an exact diagnosis.

Examinations of discharges is also an additional method. These methods usually help us to arrive at an **exact diagnosis**.

5. Rontgenological Examination.  
X-rays.

### 5. Rontgenological Examination.

Examination by X-ray is another great help to arrive at a sure diagnosis. The advantages and disadvantages, and effectiveness, or otherwise in producing different kinds of shades and opacity in different swellings are discussed elsewhere. *Vide* Regional Surgery.

6. Special Physical Examinations

### 6. Special Physical Examinations.

Special sense-organs require special methods of examinations, when such an organ is implicated by new growths. Important diagnostic points are elicited by such examinations. Examination by Tuning-Forks for the ear troubles, examination of errors of refraction by various ophthalmoscopic instruments are such instances.

## SUMMARY.

### THE DIAGNOSIS OF SWELLINGS AND GROWTHS.

1. Determine the **Anatomy** or the Site and Position.
2. Physical characters.

#### I Inspection by the eye :--

- (i) Is it actually a swelling?
- (ii) Single or Multiple?
- (iii) Deformity. Malformations.
- (iv) Shape.
- (v) Colour.
- (vi) Surface.

#### II. Palpation.

- (a) Touch by one finger and elicit :--
  - (i) Whether it pits on pressure.
  - (ii) Whether it dimples.

Whether the surface skin can be folded up.

(b) Pinch it with two fingers.

(c) Palpate with all fingers—flat, of one hand.

(i) Hot or cold?

(ii) Tender?

(iii) Crepitant?

(iv) Cracklings? Egg-shell, fine, dry.

(v) Emphysematous?

(vi) Gurgling?

(vii) Pulsating?

(d) Grasp with all fingers of one hand,—squeeze.

(i) Consistence.

(ii) Translucency.

(iii) Reducibility.

(e) Examination by both hands or Binual examination.

(i) Mobility.

(ii) Adhesion.

(iii) Fluctuation, Tension, Elasticity, Compressibility.

### III & IV. Percussion and Auscultation.

(i) Fluctuation-Wave.

(ii) Premitus.

(iii) Thrill.

(iv) Tympanitic note.

### V. Mensuration.

### VI. Clinical History.

(1) Congenital.

(2) Acquired.—Acute and chronic.

(i) Traumatic.

(ii) Inflammatory.

(iii) Neoplastic.

(iv) Obstruction.

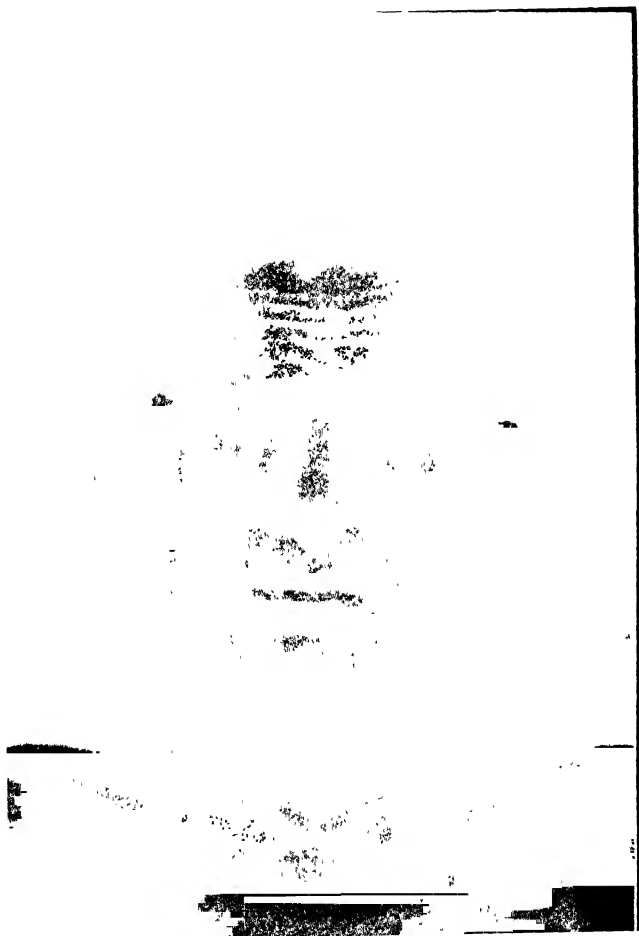
(3) Progress.

Increasing, diminishing, stationary, changes of position and direction.

## 332    DIAGNOSIS OF SWELLINGS & GROWTHS

### VII. Corroborative Examinations :—

- (i) General clinical observations age, sex, occupation, concomitant affections.
  - (ii) Diagnostic minor operations.
  - (iii) X-rays examinations.
  - (iv) Microscopical and Chemical examinations.
  - (v) Bacteriological examinations.
  - (vi) Examinations by Special Instruments for special senses.
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